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CARCINOMATOUS GASTRIC ULCER

MISLEADING RESULTS OF MEDICAL THERAPY

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It is apparent that the rank and file of the medical profession are becoming increasingly conscious of the fact that small, circumscribed, innocent looking ulcerous gastric lesions may actually be carcinomatous. Progress in our knowledge in this respect so far is due largely to disclosures by roentgenoscopy and the unexpected results of microscopic examination of presumably benign excised lesions or lesions in resected portions of the stomach.

From time to time a patient who has what is proved eventually to be a malignant circumscribed gastric lesion undergoes treatment for the usual chronic benign ulcer. There are extenuating reasons for this, because an ulcerating form of carcinoma may be indistinguishable from a benign one on the basis of the usual criteria. Moreover, the patient may refuse immediate operation irrespective of the probable nature of the lesion. Finally, the risk of the operation under certain circumstances may be greater on first blush than the risk of death from carcinoma. While any unnecessary delay in the proper treatment of a malignant growth always is to be deplored, there is the advantage of learning something of the behavior of such lesions under an intensive medical regimen.

While a considerable literature bearing on the differential diagnosis of carcinomatous and benign ulceration has accumulated, insufficient stress has been placed on the diagnostic significance of the response to medical treatment. In my judgment the roentgenologist and the surgeon have been particularly remiss in this respect. The circumspect physician is today as much concerned with the degree of demonstrable anatomic change in an ulcer undergoing treatment as with the symptomatic improvement. In fact, he has not placed too much credence on the latter for some years because he realizes that disturbances arising from gastric carcinomas of all types can be relieved temporarily in a disconcertingly large number. He may also be aware of the fact that in an average of almost a third of all verified cases of gastric carcinoma an ulcer type of complaint may be the initial or predominating one. And with much justification he regards the complete and fairly prompt disappearance of all roentgenographic evidence of a lesion and of the symptoms as trustworthy and almost absolute proof of benignancy.

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Read before the Section on Gastro-Enterology and Proctology at the Ninety-Second Annual Session of the American Medical Association, Cleveland, June 6, 1941.

However, before any hard and fast rules can be laid down, authoritative observations must be made on a number of histologically verified cases sufficiently large to be of statistical value. This applies to the effect of adequate treatment, not only on benign but on carcinomatous lesions as well. With respect to the former, we are treading on more familiar ground. Uncomplicated subacute ulcers and chronic ulcers not of too long duration usually heal without difficulty, although the latter may recur eventually. But incomplete healing of a benign ulcer due to one of the various well recognized factors obviously can give rise to difficulties in differential diagnosis. Suffice it to say that the failure of the niche to disappear completely after an adequate course of intensive treatment does not imply necessarily that the lesion is carcinomatous. Infrequently even an uncomplicated ulcer may undergo complete healing, with persistence of a niche, as shown by Unger and Poppel.[1] That this phenomenon does not occur oftener has frequently puzzled me when recalling that the bulbar deformity caused by chronic duodenal ulcers will persist almost invariably after such ulcers are healed completely and permanently. Undoubtedly the most reliable evidence as to the extent of healing of a gastric lesion is afforded by the gastroscopic examination. Our experience in this respect supports the contentions of Templeton and Schindler [2] and others that an ulcer still may be active and unhealed even though there is complete disappearance of the niche from a roentgenologic point of view.

Judging from past published reports, the large majority of carcinomatous ulcers or ulcerating carcinomas decrease little in size, if at all, under the influence of medical treatment. Some actually have been known to increase in size. Whether this is true of the earliest preinvasive types of carcinoma, so-called carcinoma in situ, especially those associated with hydrochloric acid in the gastric contents, remains to be seen. Taylor,[3] on the basis of repeated gastroscopic observations, expressed the conviction that the failure of an ulcer to change at all after three weeks of intensive treatment in the hospital is sufficient evidence of malignancy. On the other hand, if on the second examination an ulcer shows signs of uniform healing, he took the attitude that malignancy can be definitely excluded. Gutmann [4] expressed the opinion that any lesion in which there is a persistence of clinical and radiologic signs after six weeks of treatment is suggestive of carcinoma. He regarded the presence of cancer as practically certain when

1. Unger, A. S., and Poppel, M. H.: Healing of Gastric Ulcer with Persistence of Niche Roentgenographically, Am. J. Roentgenol. **39**: 592-595 (April) 1938.

2. Templeton, F. E., and Schindler, Rudolf: Roentgenologic and Gastroscopic Studies in Chronic Gastritis and Peptic Ulcer, Am. J. Roentgenol. **41**: 354-367 (March) 1939.

3. Taylor, Hermon: Practical Evaluation of Gastroscopy, Lancet **1**: 131-135 (Feb. 1) 1941.

4. Gutmann, R. A.: Ulcères bénins, ulcères transformés et cancers ulcériformes, Gastroenterologia **48**: 259-263 (Nov.) 1939.

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there are a persistence of clinical symptoms and the increase of roentgenologic signs, and as definite for carcinoma disappearance of the clinical symptoms with progressive increase of the roentgenologic signs. Mallory [5] made observations on 4 cases of early ulcerous gastric carcinoma in which there was definite diminution in the size of the crater. In the first case, in which there was an ulcer in the prepyloric region, the niche itself had disappeared completely on roentgenologic examination after eight weeks of treatment.

The subject is one of considerable significance because a fifth of all the carcinomatous lesions removed at the Mayo Clinic in recent years may be within the size range of benign ulcer (4 cm. or less in diameter). As only 6.5 per cent were small (2.5 cm. or less) MacCarty [6] felt that gastric carcinoma was not being diagnosed early.

While repeated experience in the past has taught me that a carcinomatous form of ulcer may undergo little objective change, if any, while under treatment, the improvement at other times, especially during the earlier stages of the lesion, can be so striking as to be misleading. The following cases, of which case 1 [7] and case 3 [8] have been reported previously, with illustrations, are indicative of this:

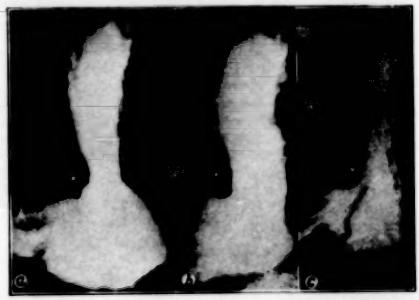

Fig. 1 (case 3).—*a*, large "penetrating" gastric ulcer, posterior wall, near lesser curvature, above incisura angularis; *b*, shallow ulcerating lesion at the original site; *c*, shallow ulcer, 2 cm. in diameter, at the original site.

REPORT OF CASES

Case 1.—A white man aged 27, who entered the Mayo Clinic on Nov. 10, 1931, for five years previously, usually late in the summer and in early spring, had experienced "spells" of indigestion lasting one to four months, with intervals of complete freedom. Two years prior to admission here he had had a severe attack during an exacerbation, strongly suggestive of a subacute "protected" perforation. On his own volition he drank milk at frequent intervals, and as a result there was considerable improvement. For nine months before he came under our observation the pain had been replaced by distress of the type of gaseous distention.

Physical examination and routine laboratory investigations were without incident. Concentration of free hydrochloric acid after an Ewald type of meal was 38. Roentgenologic examination, made Nov. 11, 1931, gave evidence of a perforating ulcer on the lesser curvature of the stomach, above the incisura angularis. A smaller one was also present just above that. After a course of intensive treatment for ulcer in the hospital, roentgen examination on Dec. 2, 1931, disclosed a complete disappearance of both lesions.

The patient was asked to return here for examination within three months but did not do so until Oct. 3, 1932, seven months after the time when he should have returned. In the meantime he had experienced the seasonal exacerbations as before. Relief by the taking of food or alkalis was less marked. He had lost 16 pounds (7.3 Kg.). Roentgen examination on Oct. 4, 1932 disclosed a large perforating ulcer on the lesser curvature, possibly a carcinoma, with a smaller ulcer just below it.

At operation on Oct. 13, 1932, a mass was found, the size of a fist, involving the middle and upper thirds of the stomach, with extensive posterior infiltration. Regional lymph nodes were enlarged. There seemed to be a 90 per cent chance that the growth was a carcinoma. The abdomen was closed after exploration only. Roentgen therapy was instituted. Progressive decline followed, with pain, ileus, vomiting and eventually signs of generalized peritonitis. The patient died on Feb. 4, 1933. At necropsy a large, ulcerating carcinoma of the stomach, with generalized peritonitis, was found.

Features of interest are the comparative youth of the patient, the association of a painful, chronic, perforated benign ulcer with a carcinoma in situ, and complete, even though temporary, disappearance of both lesions, from a roentgenographic standpoint, after treatment.

Case 2.—A white man aged 30, who entered the Mayo Clinic on Feb. 6, 1940, had always had a "weak stomach" and was an "easy vomiter." His mother "suffered from a peptic ulcer" for a number of years but died of cancer of the stomach at the age of 45 years. Since 1934 the patient had had ulcer-like manifestations, much belching and resulting gaseous distention. In June 1935 an ulcer, apparently on the lesser curvature in the prepyloric region, was found on roentgen examination. After three weeks of intensive treatment the lesion had disappeared completely on reexamination. In October 1936 there was a recurrence of the original symptoms, characterized by epigastric pain appearing one hour after each meal, which appeared to be relieved more or less completely by food and alkalis. Roentgen examination in December 1936 disclosed only antral spasm. Because of incomplete relief by treatment, operation was advised in March 1937. On exploration of the stomach an ulcer was not found but pyloroplasty was done.

Since this operation the patient has been free of his former ulcer-like discomfort, but for a year and a half prior to entering the clinic he had had much gaseous distention, nausea and vomiting, the latter sometimes of retention character, and a loss of weight. In February 1939 analysis of gastric contents after stimulation with histamine revealed a total acidity of 106 and free hydrochloric acid of 88 clinical units.

The positive findings obtained on admission were moderate anemia, alkalemia and an ulcerating lesion in the distal third of the stomach with pyloric obstruction on roentgen examination. An analysis of 350 cc. of vomited material disclosed considerable altered blood and on titration free hydrochloric acid of 30 units. At operation on Feb. 10, 1940 an annular ulcerated colloid adenocarcinoma (8 by 7 by 2 cm.), grade 4 (Broders' method), beginning just above the pylorus and completely encircling it, was removed. The overlying serosa and regional lymph nodes were involved.

The prepyloric location of the lesion in spite of the comparative youth of the patient and the history of gastric carcinoma in the patient's mother were significant. One can only speculate as to what relation the original lesion, which disappeared under treatment, bore to the ultimate extensive one.

Case 3.—A white woman aged 44, married, entered the hospital directly on July 23, 1939 because of recent hematemesis of moderate severity. For a period of four years the patient

5. Mallory, T. B.: Carcinoma in Situ of the Stomach and Its Bearing on the Histogenesis of Malignant Ulcers, Arch. Path. **30**: 348-362 (July) 1940.

6. MacCarty, W. C.: Early Cancer of the Stomach and Its Clinical Significance, Am. J. Digest. Dis. **5**: 549-554 (Nov.) 1938.

7. Rivers, A. B., and Dry, T. J.: Differentiation of Benign and Malignant Gastric Ulcers: Unreliability of Diagnostic Criteria, Arch. Surg. **30**: 702-715 (April) 1935. Eusterman, G. B., and Balfour, D. C.: The Stomach and Duodenum, Philadelphia, W. B. Saunders Company, 1935, p. 580.

8. Eusterman, G. B.: Small Carcinomatous Gastric Lesions Simulating Chronic Benign Ulcer: Present Status of Differential Diagnosis and Treatment, Minnesota Med. **23**: 703-709 (Oct.) 1940.

had had indigestion, remittent in course but progressive in severity. This was characterized by a low retrosternal pain appearing a half to one hour after each meal, often extending laterally to the left hypochondrium or posteriorly to the left subscapular region. The duration of the pain was usually an hour or two. Ingestion of warm fluids or alkaline mixtures, or induced vomiting, afforded relief rather uniformly. Roentgenoscopy in December 1937 at the patient's home by a competent roentgenologist disclosed an ulcer "quite high up in the stomach and on the posterior wall." After this examination the patient entered the hospital in her home town and submitted to treatment for ulcer for a period of five weeks. Then she enjoyed good health until March 1939, at which time there was a recurrence of her original symptoms in aggravated form, precipitated by emotional and physical stress. Four days prior to admission here the roentgenologist again confirmed the presence of the original lesion without any significant changes. The physical examination and routine laboratory investigations were without special incident. The positive findings on July 29, 1939 were moderate low grade hypochromic microcytic anemia and the presence of a large "penetrating" ulcer on the posterior wall of the stomach near the lesser curvature above the incisura angularis (fig. 1*a*). Analysis of the gastric contents (Ewald) disclosed a total acidity of 46 and free hydrochloric acid of 26 clinical units, as well as the presence of considerable altered blood. The total aspirated contents measured 160 cc.

Intensive treatment for ulcer was instituted promptly. This treatment, however, was interrupted for two weeks during August because of an intervening obscure illness characterized

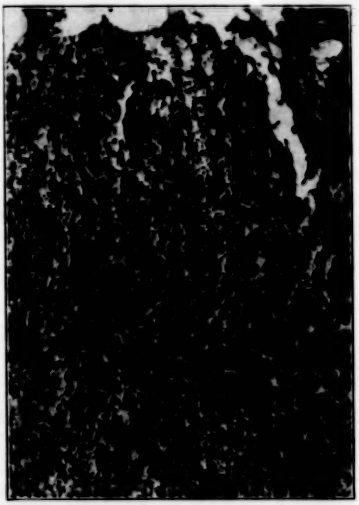

Fig. 2 (case 3).—Grade 3 adenocarcinoma in which the cells have spherical and irregular hyperchromatic nuclei, with a little tendency to acinar formation.

by fever, headache, peripheral neuritis, purpura, nausea, vomiting, icterus and leukopenia. The guaiac reaction for occult blood in the feces was intermittently positive and the benzidine reaction was usually positive throughout the period in the hospital. Roentgen examinations on Aug. 6 and Sept. 1, 1939 showed that the lesion had decreased considerably in size.

At our request the patient returned here for another examination on Dec. 8, 1939, although she not only had been free from symptoms but had gained 8 pounds (3.6 Kg.) since returning home. Roentgenoscopy at this time revealed a small shallow defect with evidence of considerable scarring and puckering of the contiguous mucosa. On gastroscopy a small ulcer on the posterior wall, just above the incisura angularis, corresponding to the site of the lesion on roentgen examination, was seen. The margin of the ulcer appeared to be quite edematous but the lesion in its major aspects appeared to be benign.

Fig. 3 (case 3).—Ulcerating adenocarcinoma, for the most part, in situ. However, there is invasion of submucosa at one point. The muscle plane is intact. (Broders.)

After another two week period of treatment, roentgen examination on Dec. 23, 1939 disclosed a shallow ulcerating lesion at the original site (fig. 1*b*). A second gastroscopy on Jan. 9, 1940 did not reveal any evidence of ulceration at this time. The gastric mucosa was scarred and puckered and there was an "area of gastritis" at the site of the previous lesion. Four days later roentgen examination revealed a shallow defect about 2 cm. in diameter at the site of the original lesion, with deformity of the surrounding gastric rugae.

On April 12, 1940 the patient again reported, as requested, for examination. She had experienced a mild recurrence of trouble for several days during the previous month as the result of emotional strain. Roentgen examination the following day disclosed findings identical with those at the last examination in January (fig. 1*c*), but on gastroscopy there was present a fairly deep ulcerating lesion the crater of which was about 1 cm. in diameter. The ulcer was irregular in contour, the border had a rolled appearance and the base was covered with a shaggy exudate. The mucosa near the incisura angularis was decidedly edematous, and several submucosal hemorrhages were seen in this area. On April 18, 1940 partial gastrectomy of the posterior Polya type was performed by Dr. Walters. The lesion proved to be an ulcerating adenocarcinoma (2 by 1.5 cm.), grade 3 (figs 2 and 3). It had invaded the submucosa but had not involved the musculature. There were associated gastritis and very early glandular involvement. A good recovery was made and the patient was enjoying good health at the time this paper was written.

Note again the evidence of almost complete healing, both from a roentgenologic and from a gastroscopic point of view. The location of the lesion on the posterior wall is significant. Can one attribute the symptoms present for a total of five years to carcinoma, or is this an example of the malignant transformation of a benign ulcer? While absolute proof is lacking, I am of the opinion that the lesion was carcinomatous from the outset, as the muscle plane was intact.

CASE 4.—A man aged 68 came to the Mayo Clinic on March 21, 1938 because of stomach trouble. In 1923 and 1928 he had experienced brief periods of gastric distress coincident with unusual nervous and physical strain. This distress, which was characterized by gas and hunger pain, was situated in the epigastrium and appeared about two hours after each meal. On relief from occupational stress the symptoms promptly dis-

appeared. The present illness began about three months prior to the patient's admission to the clinic, during the Christmas holiday (December 1937). Epigastric distress appeared about one hour after each meal and persisted for several hours. Alkalis or milk afforded relief. There was no impairment of weight, appetite or strength. In February 1938 a gastric ulcer was diagnosed after roentgen examination. Ambulant treatment for ulcer was instituted promptly and the symptoms disappeared. Because there was not a proportionate improvement in the roentgenographic appearance of the lesion, the patient was referred to the clinic for an opinion.

The general physical examination gave satisfactory results. The patient was short, stocky and well nourished. Analysis of gastric contents after an Ewald meal revealed a total acidity of 32 clinical units and free hydrochloric acid 20. The total contents removed measured 125 cc. Roentgen examination on March 28, 1938 revealed a shallow ulcer on the lesser curvature just below the incisura angularis. The presence of the ulcer was confirmed by gastroscopy three days later. The gastroscopic appearance was that of a small benign lesion on the lesser curvature and anterior wall distal to the incisura angularis.

The patient was hospitalized and underwent intensive treatment for more than a month. Roentgen examination was repeated April 18, 1938, but this failed to disclose any sign of the ulcer. On gastroscopy, July 7, 1938, the original ulcer had disappeared completely. Roentgen examination on Sept. 14, 1938 again revealed normal conditions. The patient had experienced none of his previous distress in the meantime. However, he was asked to return periodically for reexamination on account of his advanced age and the recent onset of the symptoms. On Jan. 17, 1939 there was roentgenographic evidence of a recurrence of the ulcerating lesion. Two days later gastroscopy revealed the lesion as depicted on the film. It was situated at the original site and appeared to be fairly well circumscribed, somewhat indurated and 2 cm. in diameter. On June 25, 1939 partial gastrectomy was performed.

The pathologic findings were as follows: An ulcerating colloid carcinoma, grade 3, measuring 2 by 2 by 1 cm., was situated on the anterior wall and lesser curvature of the stomach, 3 cm. above the pyloric ring. The serosal surface of the stomach immediately underlying the ulcer showed carcinomatous lymphangitis. No involvement of the lymph nodes was found.

The patient was examined again on Feb. 13, 1940 and appeared to be in excellent health. At the present writing he has no disturbances of any kind and appears to be enjoying good health.

This is a most striking example of the disappearance of all symptoms and signs of early gastric carcinoma from both the roentgenologic and gastroscopic point of view after treatment. It was largely because of the late appearance of the lesion at an advanced age that we felt close surveillance necessary in the ultimate interests of the patient. Such apprehension was justified by the course of events.

That other physicians have had similar experiences to our own is attested by the following case:

CASE 5.—A white man aged 59, who entered the Mayo Clinic Feb. 3, 1941, for fifteen years had experienced intermittently "mild heartburn" appearing an hour and a half after meals, which was relieved regularly by sodium bicarbonate. After an attack of syncope and melena in February 1939, examination elsewhere by competent observers disclosed the presence of an ulcer with a large crater on the posterior wall of the stomach. He underwent intensive treatment, and roentgen examination in May 1939 showed that all signs of the lesion had disappeared. The patient was also entirely free from symptoms. After a brief episode of moderately severe epigastric pain, which extended posteriorly, and vomiting, in August 1940 examination disclosed that the original lesion had recurred. Under a second course of intensive treatment the ulcer gradually decreased in size so that by Nov. 1, 1940 it was barely visible on roentgen examination.

Three weeks prior to admission here the symptoms were characterized by progressive decrease in gastric capacity and appetite, without appreciable loss of weight, or pain. A burning discomfort was present when the stomach was empty.

Physical examination disclosed an extensive mass in the right portion of the epigastrium, and gastric analysis showed the presence of free hydrochloric acid. Roentgen examination disclosed the presence of an extensive defect involving the lower two thirds of the stomach. At operation on Feb. 8, 1941 a huge inoperable carcinoma involving practically the entire stomach was found. There was extragastric extension into the gastrohepatic omentum, and the growth extended upward nearly to the insertion of the esophagus.

Note the large size of the crater as well as the posterior location of the original lesion, features which not only justify a healthy suspicion of carcinoma but prompt surgical intervention.

It is apparent that gastric carcinoma not only may masquerade successfully as benign ulcer but may react to treatment in similar fashion as well. In this article I have particularly tried to avoid the highly controversial subject of the causal relation of chronic benign ulcer to carcinoma. In the light of present knowledge the problem remains an insoluble one. All types of gastric cancer are notoriously prone to ulceration, the process ranging from superficial erosion to deep crateriform formation and frequently even to the extent of causing perforation. Such process has been attributed ordinarily to infection or interference with the blood supply. As regards the stomach, the factor of peptic ulceration in the presence of free acid has been given serious consideration for years by several German observers. Their views have found a recent champion in Mallory on the basis of histologic and clinical evidence and the nature of the response to treatment. The rather prompt diminution in the size of the crater and even its actual disappearance can be attributed in all probability to two factors: first, the reparative effect of treatment on secondary peptic ulceration of cancerous tissue identical with that which obtains in benign ulcer and, second, the filling in of the base of the ulcer by cancerous tissue. The latter factor was also pointed out by Rigler.[9] In Mallory's third case "the former ulcer crater was completely filled with carcinoma in situ."

The evidence already at our disposal seems to justify close scrutiny of all gastric lesions undergoing medical treatment, no matter how favorable the response, if the slightest suspicion of carcinoma exists. Such suspicion is especially justifiable under the following circumstances: location of the lesion in the prepyloric first inch, posterior wall or greater curvature, large size, consistent achlorhydria after stimulation with histamine, persistent or frequent recurrence of occult blood in the feces during treatment, late onset in an elderly person, and reduced gastric acidity and secretory volume estimated under basal conditions, especially in association with early pyloric obstruction. All patients submitted to treatment should undergo routine examination at intervals of three months for at least a year and at intervals of six months thereafter for an additional period of twelve to eighteen months.

CONCLUSIONS

Small ulcerating forms of gastric carcinoma not only may be clinically indistinguishable at times from chronic benign ulcer but may respond to medical treatment so

9. Rigler, L. G.: The Roentgen Diagnosis of Small Carcinomata of the Stomach, Wisconsin M. J. 34:236-241 (April) 1935.

favorably as to give a wrong impression of their true nature. Such response seems especially characteristic of carcinoma in situ.

The real and apparent favorable reactions to treatment of early carcinomatous ulcers are attributed, respectively, to the effect of such treatment on secondary peptic ulceration of cancerous tissue and to the filling in of the base of the ulcer with the latter.

The rate and extent of healing of an ulcerous gastric lesion undergoing adequate treatment are of primary importance in differential diagnosis. However, all treated patients should be submitted to periodic reexamination irrespective of how favorable the response. Such follow-up is particularly imperative in the presence of lesions with the clinical features which have been discussed.

BLEEDING PEPTIC ULCER

CLINICAL APPRAISAL OF VARIOUS METHODS OF TREATMENT BASED ON A SERIES OF 408 CASES

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The methods advocated in the treatment of massive hemorrhage from peptic ulcer have basically followed four general plans: (1) initial fasting and dietary restriction, (2) immediate feeding, (3) intubation and (4) surgery. Varied mortality percentages have been reported not only for the different methods but with the same procedures. Although these variations may be due to differences in the type of case encountered, nevertheless there is confusion as to the best therapeutic approach in bleeding peptic ulcer. The most commonly employed method has been the restricted dietetic regimen, based on the theory that an initial fasting period of variable duration followed by a diet of the Sippy type aids in healing the ulcer and prevents recurrence of bleeding by resting the stomach and neutralizing the acidity. The mortality with this method has ranged from 4.2 per cent in 94 cases[1] to 25 per cent in 191 cases.[2] The immediate feeding plan was first advocated in 1904 by Lenhartz,[3] who gave milk and eggs from the day of admission and scraped beef on the sixth day. He claimed that anemia and depletion were more readily overcome and that recovery was more rapid. His mortality rate was 2.1 per cent in 146 cases.[4] Andresen[5] advised the immediate feeding of gelatin mixtures and withheld venoclyses and transfusions during the initial period. He reported a mortality of 2.5 per cent in 120 cases.[6] LaDue[7] treated 79 patients with Andresen's diet and had a mortality of 6.3 per cent, including 2 patients who received parenteral fluids and succumbed. In 1933 Meulengracht[8] advocated a liberal puréed vegetable and minced meat diet from the day of admission. His mortality rate was 2 per cent in 491 cases.[9] In this country the mortality percentages with this method had been reported as 0 in 15 cases,[10] 5.3 in 133 cases[11] and 8.6 in 23 cases.[12] Duodenal intubation was suggested by Einhorn[13] in 1920. By this method he gave a liberal diet beyond the ulcer area, at times introducing the tube within six to twelve hours after admission.[14] In 1925 one of us[15] reported a series of 40 cases treated with duodenal alimentation without a fatality. Soper[16] used a nasal retention catheter, at first lavaging the stomach and then giving duodenal feedings. Woldman[17] gave a continuous colloidal aluminum hydroxide drip through a nasogastric tube inserted just beyond the cardia and permitted a bland diet by mouth. His mortality rate was 2 per cent in 144 cases. Surgical intervention has been attended by a mortality rate ranging from 5 to 30 per cent. Finsterer[18] advised immediate resection in acute hemorrhage, while Gordon-Taylor,[19] Allen and Welch[20] and Hinton[21] advocated surgical intervention in selected cases, and Blackford and Williams[22] urged prompt surgery in the older age group.

In an attempt to evaluate some of the methods employed, a series of 408 patients with bleeding peptic ulcer, treated in the various divisions of two hospitals from 1927 to April 1941, was studied. They comprised ward as well as private patients. Three hundred and forty-five were males and 63 were females. The youngest was 12 years of age and the oldest 86 years. Three were Negroes. Two hundred and seventy were under 50 years of age; of these 13 died, a mortality of 4.8 per cent. One hundred and thirty-eight patients were 50 years of age or over; of these, 23 died, a mortality of 16.6 per cent. In 18 of the 408 cases, or 4.4 per cent, hemorrhage was the sole cause of death. In 18 other cases, or 4.4 per cent, complicating diseases contributed to the fatalities. The total mortality for the series was 8.8 per cent. In table 1 the age and sex distribution and mortality have been outlined by decades.

A duodenal ulcer was demonstrated in 287 cases, a gastric ulcer in 55, both gastric and duodenal ulcers in 6, a marginal ulcer in 18, and both a marginal and a duodenal ulcer in 1. In 22 cases the radiologic examination after the hemorrhage was negative, but a sub-

From the Lenox Hill and Beth Israel hospitals.

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1. Manheim, S. D.: The Incidence and Prognosis of Hemorrhage as a Complication of Gastroduodenal Ulcer, M. J. & Rec. **124**: 98, 1926.
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3. Lenhartz, H.: Eine neue Behandlung des Ulcus ventriculi, Deutsche med. Wchnschr. **30**: 412, 1904.
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9. Meulengracht, E.: The Medical Treatment of Peptic Ulcer and Its Complications, Brit. M. J. **2**: 321 (Aug. 12) 1939.
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14. Einhorn, Max: Management of Acute Hemorrhages in Peptic Ulcers, M. J. & Rec. **135**: 219 (March 2) 1932.
15. Rafsky, H. A.: The Treatment of Gastric Hemorrhage in Peptic Ulcer, Internat. J. Med. & Surg. **38**: 8, 1925.
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17. Woldman, E. E.: The Treatment of Massive Gastroduodenal Hemorrhage by the Continuous Administration of Colloidal Aluminum Hydroxide, Am. J. Digest. Dis. **8**: 39 (Feb.) 1941.
18. Finsterer, H.: Die Bedeutung der Resektion zür Ausschaltung für die Behandlung des nicht Resezierbaren Ulcus duodeni, Wien. klin. Wchnschr. **46**: 545 (May 5) 1933.
19. Gordon-Taylor, G.: Attitude of Surgery to Hematemesis, Lancet **2**: 168, 1927.
20. Allen, A. W., and Welch, C. E.: Peptic Ulcer Considered from Surgical Point of View, New England J. Med. **220**: 103 (Jan. 19) 1939.
21. Hinton, J. W.: Operation for Massive Hemorrhage in Peptic Ulcer, Ann. Surg. **110**: 376, 1939.
22. Blackford, J. M., and Williams, R. H.: Fatal Hemorrhage from Peptic Ulcer, J. A. M. A. **115**: 1774 (Nov. 21) 1940.

sequent examination showed a duodenal ulcer in 3 of these and a marginal ulcer in 3 others. In addition, gastroscopy revealed a marginal ulcer in 2 and an erosive gastritis in 2 others. In the remaining 12 cases and in 19 others in which further examinations were refused the previous history and the clinical course clearly indicated the presence of a peptic ulcer.

TABLE 1.—*Age and Sex Distribution and Mortality by Decades*

Age in Years	Male	Female	Total	Deaths	Per Cent Mortality
10-19	9	2	11	0	0
20-29	61	7	68	3	4.4
30-39	81	15	96	7	7.3
40-49	82	13	95	3	3.2
50-59	69	13	82	7	8.5
60-69	36	12	48	13	27.1
70-79	6	1	7	2	28.6
80-89	1	0	1	1	100
Total	345	63	408	36	8.8

271 patients, or 11 per cent, died; of these, 14, or 5.2 per cent, died of bleeding per se, while 16, or 5.8 per cent, had complicating diseases in addition to the hemorrhage.

2. Thirty-nine patients were given the Meulengracht diet immediately on admission to the hospital. In 26 of these this plan could be followed without interruption. It had to be discontinued in 13, owing to a recurrence of bleeding in 7, 3 of whom died; severe pain in 3, 1 of whom had a perforation of his ulcer and died postoperatively, and severe nausea and vomiting in 3. Four of the 39 patients, or 10.3 per cent, died; of these, 2, or 5.1 per cent, died of bleeding per se.

3. Thirty-four patients were treated by duodenal alimentation according to Einhorn's method. The tube was inserted within twenty-four hours after admission. Small oral feedings of milk were given hourly until the tube reached the duodenum, when duodenal alimentation was instituted. One of the 34 patients, or 2.9 per cent, died of exsanguination.

TABLE 2.—*Analysis of Mortality According to Severity of Bleeding and Plan of Treatment*

Grade of Bleeding*	Restricted			Meulengracht			Duodenal Intubation			Combined			Total		
	Cases	Deaths	Per Cent Mortality	Cases	Deaths	Per Cent Mortality	Cases	Deaths	Per Cent Mortality	Cases	Deaths	Per Cent Mortality	Cases	Deaths	Per Cent Mortality
1	48	1	2.1	12	1	8.5	10	0	0	14	0	0	84	2 0†	2.4 0†
2	87	6	6.9	9	0	0	13	0	0	17	0	0	126	6 0†	4.8 0†
3	56	5	8.9	14	1	7.1	7	0	0	13	0	0	90	7 4†	7.8 4.4†
4	80	18	22.5	4	2	50.0	4	1	25	20	1	5	108	21 14†	19.4 13†
Total	271	30 14†	11 5.2†	39	4 2†	10.3 5.1†	34	1†	2.9†	64	1†	1.6†	408	36 18†	8.8 4.4†

	Hemoglobin	Red Blood Cells per Cu. Mm.
* Grade 1	12 Gm. (80%) or above	4,000,000 or above
Grade 2	9-11.8 Gm. (60-79%)	3,000,000-3,990,000
Grade 3	6.8-8.8 Gm. (45-59%)	2,250,000-2,990,000
Grade 4	Below 6.8 Gm. (below 45%)	Below 2,250,000

† Mortality from bleeding per se, excluding cases with complicating diseases.

Frank hematemesis alone was present in 71 cases, tarry stools alone in 160 and both hematemesis and melena in 177. Patients with only blood streaked vomitus or occult blood in the stool were not included in this series. Two hundred and eighty-three patients were seen during their first hemorrhage, 84 in the second and 25 in the third. Sixteen had four or more hemorrhages. One patient was seen in his twelfth bleeding episode. In 43 cases hemorrhage was the first symptom to appear.

The severity of the bleeding in this series was graded according to the lowest recorded hemoglobin value and red blood count in each case, grossly as indicated in table 2. Where there was overlapping of the values into two grades, the final classification was based on additional data such as the presence of restlessness, vertigo, syncope or collapse, the pulse rate, blood pressure, serum proteins and blood urea nitrogen and the amount of bleeding actually observed during the hospital stay. These criteria are a more satisfactory index of the degree of bleeding than the use of such terms as profuse, gross, massive or copious hemorrhage, which may be variously interpreted.

The patients in this series were further classified according to the method of treatment, as follows:

1. Two hundred and seventy-one patients were treated by an initial period of fasting of variable duration followed by a Sippy type of diet. Thirty of the

4. Sixty-four patients were treated by a combined method. On admission to the hospital food was withheld for one day, or longer if bleeding continued. During this time venoclyses of 5 per cent dextrose in physiologic solution of sodium chloride to which were added 300 mg. of ascorbic acid and 25 mg. of thiamine hydrochloride were slowly administered. When necessary, indirect transfusions of 250 to 500 cc. of citrated blood were slowly given. If whole blood was not available, serum or plasma was used. The daily fluid intake during this period was limited to about 3 per cent of the body weight. When bleeding had ceased, as evidenced by a stabilized blood pressure, pulse, blood findings and the clinical impression, hourly feedings of 1 ounce (30 cc.) or more of whole milk were given for one day or longer. If well tolerated, an ounce or more of cereal was given alternately with the milk every half hour for one or two days. During this time the fluid intake was supplemented by proctoclyses or venoclyses if necessary. Thirty-three patients in this combined group, who had no pain, had the feedings of milk and cereal followed, on the fourth to the seventh day after admission, by a diet consisting of cereals, cream soups, bread and butter, custards, jello, junket, milk, raw and soft boiled eggs, baked and mashed potatoes and puréed bland vegetables. These foods were given in increasing amounts for five days, when broiled white fish, minced chicken, lamb and scraped beef were added.

At this time no restriction was placed on the patient's appetite. Thirty-one patients in this combined group who were in pain had a duodenal tube inserted on the fourth to the seventh day after admission. When the tube reached the second portion of the duodenum, feedings consisting of one egg, 8 ounces (15 cc.) of milk and 1 tablespoon of lactose were given every two hours through the tube, as well as 500 cc. of a 5 per cent dextrose solution or of tap water, twice daily. The total daily intake was about 2,400 calories. The duodenal feedings were continued for two weeks, after which a liberal diet was given by mouth. The average period of bed rest was three weeks and the total hospital stay about four weeks. Of the 64 patients, 1 died of exsanguination. She was treated by a combination of initial restriction and duodenal alimentation. The mortality for this group was 1.6 per cent.

Morphine and the barbiturates were administered as indicated to insure mental and physical rest. Alkaline powders, similar to the original Sippy formulas, or colloidal aluminum hydroxide preparations, or the Meulengracht powder with ferrous lactate were employed. Iron preparations were prescribed as indicated, as were thiamine hydrochloride, ascorbic acid and menadione.

The experience with the four methods of treatment has been analyzed according to comparable grades of bleeding in table 2 and in the age groups above and below 50 years in table 3. In both tables, the influence of complicating diseases on the mortality is shown.

Twenty-eight of the 408 patients included in this series came to operation after a period of medical treatment varying from one day to three weeks. Twenty-three were originally treated with the restricted dietetic regimen. Four of these died postoperatively. One was treated by the Meulengracht diet and died following an operation for a perforation. Two patients were originally treated by duodenal intubation and 2 by the combined method. All 4 survived surgery. Five of the patients who came to operation had grade 1 bleeding, 9 grade 2, 4 grade 3 and 10 grade 4. In table 4 the mortality percentages of medical treatment alone and of both medical and surgical treatment above and below 50 years of age are shown.

There were 3 patients who were treated with Andresen's diet and 6 with a continuous intragastric drip of colloidal aluminum hydroxide. These were not included in this series because they were too few in number to permit any conclusions.

CONTRIBUTORY FACTORS

The factors which played a role in precipitating the hemorrhages in this series were, in the order of frequency, infections of the upper part of the respiratory tract, alcoholic beverages, emotional disturbances, dietary indiscretions, physical exertion and overwork. Trauma to the abdomen was stated to have occurred in 1 case. The greatest number of bleeding episodes occurred from the latter part of October to the early part of March, which corresponds to the increased incidence of respiratory infections. Bleeding followed intoxication in a few cases but more frequently it occurred in those in which small amounts of alcohol were taken with meals. Emotional strain manifested itself mainly in worry over financial matters, business or profession, family illness and domestic difficulties.

ANALYSIS OF MORTALITY

Thirty-six patients in this series died. There were 29 men and 7 women. The youngest was 22 years old and the oldest 86, the average being 53 years. The duration of their symptoms varied from three to thirty years, the average being five and six-tenths years. In 3 cases the onset of the fatal hemorrhage was the first symptom. Of the 36 patients, 23 died during their first hemorrhage, 10 during their second, 2 during their third and 1 during his sixth hemorrhage. Thirteen had a gastric ulcer, 20 a duodenal ulcer, 2 both gastric and duodenal ulcers and 1 a hemorrhagic gastritis of a resection stump.

TABLE 3.—*Analysis of Mortality According to Age Groups and Plan of Treatment*

	Restricted			Meulengracht			Duodenal Intubation			Combined			Total		
Age	Cases	Deaths	Per Cent Mortality	Cases	Deaths	Per Cent Mortality	Cases	Deaths	Per Cent Mortality	Cases	Deaths	Per Cent Mortality	Cases	Deaths	Per Cent Mortality
Below 50 yr.	162	11	6.6	30	1	3.3	29	0	0	49	1	2.1	270	13 8*	4.8 2.9*
50 yr. and above	109	19	17.4	9	3	33.3	5	1	20	15	0	0	138	23 10*	16.6 7.2*
Total	271	30 14*	11 5.2*	39	4 2*	10.3 5.1*	34	1*	2.9*	64	1*	1.6*	408	36 18*	8.8 4.4*

* Mortality from bleeding per se, excluding cases with complicating diseases.

Eighteen of the 36 patients who died, or 4.4 per cent of the total, had other diseases in addition to the bleeding ulcer which contributed to the fatal outcome. Of these 18 patients, 8 had grades 1 and 2 bleeding, of whom 7 were over 50 years of age. The remaining 10, of whom 6 were 50 years of age or over, had grades 3 and 4 bleeding. The complicating diseases were cerebral accidents, coronary thrombosis, congestive heart failure, renal disease, perforation of the ulcer, adenocarcinoma of the rectum and bronchopneumonia.

TABLE 4.—*Comparison of Mortality Above and Below 50 Years of Age on Medical and Both Medical and Surgical Treatment*

	Medical			Medical and Surgical		
Age	Cases	Deaths	Per Cent Mortality	Cases	Deaths	Per Cent Mortality
Below 50 years....	252	10	3.9	18	3	16.6
Above 50 years...	128	21	16.4	10	2	20.0
Total.....	380	31	8.2	28	5	17.9

Of the 36 patients who died, 26 received transfusions, which were direct in all but 1 instance. Bleeding was not the sole cause of death of 11 of these 26 patients. The mortality rate in the entire series increased with the severity of the hemorrhage, whether transfusions were given or withheld. As the severity of the bleeding increased, a greater proportion of the patients received transfusions; 76.5 per cent of the 170 patients who

received transfusions, as compared to 28.6 per cent of the 238 patients who received no transfusions, had grades 3 and 4 bleeding. The patients in this series who received no transfusions were chiefly those who had a single hemorrhage without recurrence of bleeding during their hospital stay; a lower mortality is to be expected in these cases. On the other hand, 3 patients with grade 4 bleeding received five, seven and eight transfusions respectively and survived. It was difficult in many cases of recurrent bleeding to avoid the conclusion that transfusion was an important factor in the survival of the patient. On the other hand, with an exsanguinating hemorrhage or a complicating disease a fatal outcome could not be charged to transfusions.

In view of the statement which recurs frequently in the literature that transfusions increase the mortality by raising the blood pressure and dislodging the clot, it was thought of interest to compare the mortality rate of patients with and without hypertension. In this series there were 42 patients whose blood pressures on admission ranged from 150 mm. to 235 mm. of mercury. Based on Allen's [23] statement that a systolic blood pressure of 140 is a hypertensive level, 26 patients whose blood pressures ranged from 140 to 150 were included in this group, making a total of 68 patients, or 16.6 per cent of the series, who had hypertension. One may assume that the usual blood pressure of these patients was higher than that recorded during the hemorrhage. Of the 68 patients, 8, or 11.8 per cent, died, as compared to a mortality of 8.2 per cent in patients without hypertension. Of the 8 patients with hypertension who died, 4, or 5.9 per cent, died of exsanguination, as compared to 4.1 per cent of patients without hypertension, in whom bleeding was the sole cause of death.

COMMENT

Quite apart from the therapeutic procedure employed, the mortality in a series of cases of bleeding peptic ulcer is influenced by three factors: the severity of the bleeding, the age distribution and the presence of other organic diseases which determine or contribute to an unfavorable outcome. Differences in these factors probably account for the varied mortality statistics presented by many authors.

The need for grading the bleeding as an index of severity is emphasized by the demonstration that the ingestion of 50 to 80 cc. of blood is sufficient to produce a tarry stool.[24] The persistence of a tarry stool or of occult blood does not in itself indicate continued bleeding, because the former often persists for several days and the latter for one or two weeks after the hemorrhage has ceased. It is generally recognized that the mortality in bleeding peptic ulcer is higher in the older age group, as the result of arteriosclerosis and complicating diseases which may result in an unfavorable outcome even with a mild hemorrhage. Forty-eight and eight-tenths per cent of the patients in this series had grades 3 and 4 bleeding with a mortality of 14.1 per cent, as compared to a mortality of 3.8 per cent in those with grades 1 and 2 bleeding. Of the patients in this series 38.8 per cent were 50 years of age or over with a mortality of 16.6 per cent, as compared to a mortality of 4.8 per cent in those under 50 years of age. Fifty per cent of the fatalities were of patients who had complicating diseases. There are certain complications which may make a fatal outcome inevitable but there are others, such as circulatory failure, azotemia and perforation, which may in part if not entirely be the result of the therapeutic procedure employed.

While mild uncomplicated cases do not, as a rule, present any problem, the treatment of severe gastroduodenal hemorrhage involves the following objectives: to aid in the arrest of bleeding, overcome shock and dehydration, combat anemia and depletion and prevent recurrence of hemorrhage. In the presence of continued bleeding, mental and physical rest are essential. The immediate need in these cases is not for food but usually for the restoration of the water balance to prevent or overcome shock, dehydration and extrarenal azotemia. If the daily parenteral fluid intake is kept to about 3 per cent of the body weight and administered slowly, there is little or no danger of increasing blood volume beyond its normal range. The regulation of the water balance is essential to the maintenance of normal function of the kidneys, which are burdened by an increased urea production at a time when the circulating blood volume is decreased. The resultant extrarenal azotemia is not proportional to the anemia,[25] and persistently high urea nitrogen levels indicate a bad prognosis, a fact which is corroborated by our experience in this study. Jones,[26] in analyzing a series of 39 fatalities, found that dehydration was an important factor in 18, and that 7 persons who had uremic blood levels did not die of anemia. Small citrated transfusions, frequently given, should be employed if the anemia is severe. The contention that transfusions raise the blood pressure, thereby dislodging the clot, cannot be directed to slowly administered drip transfusions, for Jones [27] has shown that transfusions of this type given in amounts up to 2,650 cc. daily produce no rise in blood pressure.

Our experience has been that progress was more satisfactory with food restriction until the active bleeding stopped. It was deemed advisable in all cases to withhold food for at least twenty-four hours after admission until it was determined that hemorrhage had ceased, at which time food was permitted in small amounts. Melena or occult blood in the stool was not regarded as a contraindication to food. If the small feedings were well tolerated, the diet was quickly increased. For patients without pain, a liberal diet was given by mouth. For those with pain, the increase in diet was accomplished by duodenal alimentation.

Meulengracht [9] has stated that food in the stomach does not increase or protract the bleeding. In this connection it is of interest to note that he was inclined to think that the bleeding had stopped in most of his cases before arrival at the hospital, and that on the whole they were of a milder character than the hospital cases in England. Five of the ten fatalities in his series of 491 cases were due to bleeding which recurred from two to nineteen days after admission. In our series of 39 patients treated with a Meulengracht diet from the time of admission, 7 patients, 3 of whom died, had a recurrence of bleeding on the second to the eighth day. In addition, 3 patients experienced severe pain, and 1 of these died of a perforation. Meulengracht did not cite the age distribution of his patients.

23. Allen, E. V.: Medical Aspects of Arterial Hypertension, Bull. New York Acad. Med. **17**: 174 (March) 1941.

24. Daniel, W. A. Jr., and Egan, Sherman: The Quantity of Blood Required to Produce a Tarry Stool, J. A. M. A. **113**: 2232 (Dec. 16) 1939.

25. Rafsky, H. A., and Weingarten, Michael: The Clinical Significance of Prerenal Azotemia in Digestive Tract Disease, New York State J. Med. **39**: 1086 (June 1) 1939.

26. Jones, F. A.: Hematemesis and Melena: Observations on Salt and Water Requirements, Brit. M. J. **2**: 332 (Aug. 12) 1939.

27. Jones, F. A.: Hematemesis and Melena: Observations on the Use of Continuous Drip Transfusions, Brit. M. J. **1**: 915 (May 6) 1939.

The decision as to surgical intervention in bleeding peptic ulcer is a difficult one. The gastroenterologist hesitates to advise surgery because medical measures alone are frequently successful even when the situation appears critical. The decision cannot be made in advance based on statistical data alone but should be made in each case by weighing the severity of the bleeding, the ability of the patient to withstand radical surgery, the presence of complicating diseases and the skill of the surgeon. We have adopted the following procedure: If bleeding continues notwithstanding medical measures, the surgical division is consulted and the patient observed by both divisions. If after the use of transfusions the systolic blood pressure remains at 90 or below, the pulse rate 130 or above and the hemoglobin 35 per cent or below, surgery is advised in the absence of a complicating disease which would render operation futile.

Another problem which frequently arises is whether to advise an interval operation to prevent a recurrence of hemorrhage. In this series 64 per cent of the fatalities resulted from the first hemorrhage. One should be guided in making a decision as to an interval operation by the ability and willingness of the patient to avoid the contributory factors previously mentioned and the presence or absence of complicating diseases. It must be borne in mind that surgery does not always succeed in preventing a recurrence of hemorrhage. Thirty-nine of the patients in this series had had a gastroenterostomy prior to their bleeding episode. Three had hemorrhages in from one to three years following a subtotal resection, two of which were from anastomotic ulcers and one from a diffuse gastritis of the stump which resulted in fatal bleeding.

Gastroscopy may be helpful in the interval between hemorrhages in determining the source of bleeding. In this series, gastroscopy revealed that 2 patients had an erosive gastritis and that 2 others had marginal ulcers which were not detected by other means.

SUMMARY

A comparative study of various methods of treating bleeding peptic ulcer was made in a series of 408 patients. A basis for grading the severity of the bleeding was evolved and the influence of age and of complicating diseases on mortality determined. The data obtained in this study revealed the following:

1. Patients with mild uncomplicated hemorrhage progressed favorably with any of the medical methods.

2. Fifty per cent of the fatalities were of patients with complicating diseases; the latter accounted for all deaths of patients with grades 1 and 2 bleeding.

3. The total mortality rate below 50 years of age was 4.8 per cent, as compared to 16.6 per cent for patients 50 years of age or over.

4. Below 50 years of age the mortality rate for surgically treated patients was 16.6 per cent, as compared to 3.9 per cent on medical measures alone; while above 50 years of age the respective mortality percentages were 20 and 16.4.

5. The mortality rate with the restricted dietetic regimen was 11 per cent, of which 5.2 per cent were due to bleeding per se; with the Meulengracht diet, 10.3 per cent, of which 5.1 per cent were due to bleeding per se; with duodenal intubation, 2.9 per cent and with the combined method herein described, 1.6 per cent; in the latter 2 instances the fatalities were due to exsanguination.

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ABSTRACT OF DISCUSSION

ON PAPERS OF DR. EUSTERMAN AND DRS. RAFSKY AND WEINGARTEN

DR. NATHANIEL E. REICH, Brooklyn: Until such time as definite factors responsible for the formation of peptic ulcers are discovered, the treatment must be empirical and symptomatic. Treatment of hemorrhage presents four problems: 1. Stop the hemorrhage. 2. Keep the patient from dying from shock or circulatory failure. 3. Treat the ulcer. 4. Treat the anemia. In stopping the immediate hemorrhage, morphine is the drug of choice. It has been objected that morphine frequently excites nausea and vomiting. However, much of the recent experimental evidence indicates that this drug produces a fall in tone, a decrease in amplitude of rhythmic contraction and a decrease in the work performed by the stomach. I have found that therapeutic doses exhibit a definite inhibition of the fasting gastric secretion in the majority of instances. In the small percentage of patients that retch constantly, it may be advisable to wash the stomach rapidly with hot saline solution. Besides stimulating clot formation, this washing also gives direct information concerning the continuation or the recurrence of bleeding. Most physicians are in agreement that the danger of increasing hemorrhage by raising the blood pressure by transfusion is extremely slight if it is given by the slow, intravenous drip method. Jones has given as much as 1,200 cc. by this method, with no evidence of continued bleeding. I have found similar results. Besides replacing blood volume, proteins and hemoglobin, transfusion has the additional advantages of acting as a powerful hemostatic and hemopoietic agent. At least 80 per cent of the lost blood should be replaced by whole blood or blood plasma. Indications for blood transfusion are: 1. Systolic blood pressure of less than 75 mm. 2. Pulse rate of more than 140 per minute. 3. Hemoglobin of less than 35 per cent. 4. Shock, delirium, persistent headache and restlessness. An excellent consideration of medical treatments of the ulcer has already been mentioned by Dr. Rafsky. Anemia is best treated with oral iron preparations. The Meulengracht diet produces a more rapid regeneration of red blood cells and hemoglobin. Small transfusions may be found advisable about one week after the hemorrhage becomes quiescent.

DR. JOHN DAY GARVIN, Pittsburgh: I will confine my discussion of Dr. Eusterman's paper to presentation of 2 cases. A woman of 60 with a vague dyspeptic history of six weeks' duration presented in a roentgenogram a very small ulcerating lesion on the lesser curvature. I initiated an ambulant regimen. Within six weeks there was a great increase in the size of the lesion. The fact that I was dealing with a malignant ulceration was now apparent. At operation, resection was done with fair facility. The pathologic diagnosis locally, and ultimately by Broders, was that of a grade 4 carcinoma. After fourteen years the resected stomach seems perfectly normal and the patient insists that she is "just fine." In this case medical therapy was not only not misleading but, indeed, furnished the diagnostic base for the proper aggressive action. The second patient is a woman of 55 with a typical ulcer history of five years' duration. As the roentgen appearance was that of a large ulcerating lesion on the greater curvature, I insisted on prompt surgery. She would not submit to an operation and was placed on an ambulant ulcer regimen. She got complete relief from all discomfort, and the roentgenologic picture changed decidedly. The ulcer niche decreased persistently in size, ultimately almost disappearing. I did not see her for five months, at which time the picture was much as before, save for an increase in the constricting nature of the lesion. She consulted another doctor who was quite certain of the benign nature of the "ulcer" and utilized a four weeks intensive hospitalization regimen. After this he too suggested surgery. The surgeon, a competent one, told me that it was definitely a benign ulcer. The pathologic diagnosis was adenocarcinoma. My colleague was completely misled by the results of medical therapy. Subjectively they were, indeed, all that could be asked. As has been said by Dr. Eusterman, the niche of a supposed gastric ulcer may diminish in size or even disappear roentgenologically under nonoperative treatment but may still turn out to be malignant, the improvement being caused by the subsiding of an inflammatory reaction associated with the malignant ulcer.

DR. JAMES F. WEIR, Rochester, Minn.: In any discussion on peptic ulcer it is necessary to distinguish gastric from duodenal ulcer. The diagnosis usually is accomplished by roentgenologic examination. Because the roentgenologic diagnosis of gastric ulcer depends primarily on the demonstration of a crater, the progress of healing can be followed readily. However, because the roentgenologic diagnosis of duodenal ulcer depends on a persistent deformity of the cap, craters are demonstrable in only 20 to 40 per cent of the cases, and because this deformity usually persists after healing there remains no satisfactory roentgenologic finding by which the activity, inactivity or healing state of duodenal ulcer can be determined. Gastric ulcers usually can be visualized through the gastroscope, but duodenal ulcers are beyond the reach of this instrument. In the medical treatment of gastric ulcer, certain principles are well established and many gastric ulcers can be shown to fulfil the requirements. Failure to do this is not necessarily a sign that the ulcers are malignant but may indicate chronic induration, a deep crater, walled off perforation or hourglass contraction, and on such evidence the ulcers may be classed as intractable and requiring surgical treatment. The problem of duodenal ulcer is somewhat different. Experience at the Mayo Clinic is similar to that of others; namely, that 75 to 80 per cent of these patients usually can be handled satisfactorily by medical measures, malignant changes practically never occur and in cases in which acute perforation, obstruction and repeated hemorrhages occur treatment is definitely surgical. In addition to these surgical cases there remains a group of cases in which severe symptoms, such as long duration of symptoms, progression in frequency and duration of attacks, lack of remissions, increasing severity of pain, nocturnal awakening, posterior extension of pain, lessening relief from food and failure to respond satisfactorily to medical treatment, appear. These symptoms usually mean induration, chronic penetration or beginning obstruction, for which surgical treatment should be instituted before the patient becomes debilitated or demoralized by pain or economic stress.

DR. SARA M. JORDAN, Boston: Dr. Eusterman's paper revives the most important problem in gastroenterology. No one who knows the statistics of incidence of cancer of the stomach can fail to be impressed by them. A recent review of our cases of gastric ulcer leads us to the following conclusions: To a confirmation of certain points in which we have believed for years; namely, that neither the size of the ulcer nor the age of the patient nor the presence of normal acid or hyperchlorhydria should lessen our suspicion of carcinoma, for some of our largest lesions have been benign and some of the smallest malignant, and malignant lesions occur often enough in the young, and benign ulcers often enough in the middle aged and old, and acid is present often enough where the lesion is malignant, so that these three criteria of size of ulcer, age of patient and presence or absence of acid have no actual practical value in the diagnosis of the individual patient, even though accumulated data on these points show a statistical trend. To a feeling of confirmation of the policy which we have practiced for many years, namely that the three criteria of complete disappearance of occult blood, x-ray defect and symptoms, are proof of healing. I would add recurrence as an indication for resection and shall not be unhappy if we have many benign lesions resected, for in the present fortunate era of almost perfected gastric resection the danger from the mortality of resection is less than the danger of gastric cancer as a sequela of recurrent gastric ulcer. Two other points are: (*a*) We believe that all greater curvature lesions are malignant. (*b*) Posterior wall ulcers, either of the median or of the prepyloric area, because often embedded in adjacent organs, cannot heal as readily as those which are not so embedded, nor can their healing be as well visualized fluoroscopically or gastroscopically. Hence, since many of the prepyloric lesions are on the posterior wall, their differentiation is more difficult and, if allowed to continue unhealed, a malignant condition may result. Even with perfected surgery it is (1) unnecessary to resect all gastric ulcers, (2) essential that to the former three criteria for benignity a fourth be added, namely a failure to recur, and (3) necessary to exercise the greatest care to make certain that complete healing has occurred and that, where there is doubt because of recurrent occult blood or of difficulty in following the lesion to complete healing because of the location of the ulcer or the management of the patient, resection should be done. There should be a careful evaluation of the behavior of the stomach wall adjacent to the ulcer, in addition to the gastroscopic appearance of the mucosa and the closing of the ulcer.

DR. SIDNEY A. PORTIS, Chicago: Increasing clinical experience has not deterred me from my original position because I have not seen any evidence presented to make a definite differential diagnosis preoperatively in a questionable lesion. Even with the gastroscope no clear differentiation can be made. I cannot understand how a gastroscopist can be sure that a given lesion is benign when a surgeon with equally good clinical experience, the abdomen open, the lesion in his hand, cannot make a final diagnosis. Therefore, as I maintained years ago and still maintain, I would trust only the microscope in making this fine differential diagnosis. I still feel that gastric lesions, for the most part, are better handled by the surgeon. I am not discussing persons in their early twenties with a small, acute ulcer, but particularly that group of patients beyond 35 years of age who are good operative risks. I am convinced that the mortality and morbidity which would accrue in handling these early lesions surgically would be far less than a similar large series of cases handled medically. I think Dr. Eusterman will recall a case that I sent up to the clinic for review, in which the gastroscopist made a definite diagnosis of carcinoma with achylorhydria. Exploration was performed and a resection of the lesion failed to reveal any evidence of malignancy. With regard to Drs. Rafsky and Weingarten's statistics, I think we ought to be careful about the evaluation of the morbidity and mortality that accrue from surgical intervention in bleeding ulcers. I do not think we are quite fair in comparing our results with others because, for the most part, these patients have surgical intervention when procrastination and medical measures have long since failed. I feel that patients who come to our attention with exsanguinating hemorrhages are better handled at the outset by surgeons than to go through the trials and tribulations of medical management. When we are able to evaluate our statistics from both sides of this problem, namely those treated early from the standpoint of surgery and those from the standpoint of medical management, we shall be in a better position to say which would be the better type of treatment. It is interesting to note how frequently the findings at operation will impress on the mind of the medical man the futility of continued medical treatment in these complicated ulcer cases.

DR. J. SHELTON HORSLEY, Richmond, Va.: We have come a long way since thirty years ago when the late Dr. John B. Deaver in his dramatic manner said "the only cure for peptic ulcer was the aseptic scalpel." We now know that a young man who has had an aseptic scalpel applied to him for a peptic ulcer of the duodenum in the way of a gastroenterostomy and has high gastric acid is in a very unhappy situation. The great majority of peptic ulcers of the duodenum are better treated by medical means. And by "medical means" I imply that the treatment of the patient is more important than the treatment of the disease. It is a problem of proper living in a quiet environment, just as much as, if not more than, diet and drugs. In the treatment of hemorrhage we have also changed a good deal. Surgeons and medical men are not as antagonistic as they used to be. The statistics of Drs. Rafsky and Weingarten would doubtless show even more strikingly the difference in the mortality of certain groups if they had put his age limit down to 40, for instance. Over 40 or even 35 years of age the mortality of hemorrhage from a peptic ulcer increases alarmingly, and the need for surgical intervention is very much greater. No matter what the blood count is, within reasonable limits, if the blood pressure is too low for too long the patient may get in such a state of shock that even transfusions of plasma or of blood cannot save. Dr. Eusterman has given, as usual, a splendid paper. There have been many cases of what appeared to be benign ulcer that have proved to be malignant. They are more important than cases of what appeared to be malignant ulcers that were really benign. One of the most important things we can bring out here is the fact that Dr. Eusterman and others have emphasized: In practically every early case of gastric cancer, medical treatment for ulcer will relieve the clinical symptoms for a time. This fact has been thought to be a therapeutic test for showing a benign lesion, just as giving quinine is for malaria. This is a common cause of delayed diagnosis

and treatment. If operation for a peptic ulcer is indicated, a partial gastrectomy should, as a rule, be done. The indirect or halfway procedures, such as pyloroplasty and gastroenterostomy, are often unsatisfactory.

DR. EVERETT D. KIEFER, Boston: If we cannot treat the condition satisfactorily our only answer to the problem is possible prevention, and that means that it is important for both the physician and the patient to realize that ulcer is an intermittent disease and a progressive disease, and that with each attack there is added damage to tissue, increased scar formation, and if this is allowed to accumulate over a period of years we get the intractable peptic ulcer; therefore it is important for the patient to establish himself on a regimen for the control of this disease. We do not speak of cure; we speak of continued control, which means a constant, a permanent regimen that will keep him free of symptoms at all times, and it is best when it is controlled by the physician by occasional examinations to show that there is not accumulating additional anatomic change in the duodenum.

DR. HENRY A. RAFSKY, New York: We found that in the initial stage the immediate problem was the restoration of the water balance. If anemia was present citrated transfusions were employed, but in this connection it should be borne in mind that the criteria by which anemia is judged may be deceptive in the early stage. Lyon and Brenner made a comparative study of the regeneration of blood in patients treated with Meulengracht and Sippy plans respectively and found no difference in blood regeneration with either method. With reference to a systolic pressure of 75 mm. of mercury, we think that this is a dangerously low level to wait for. If the blood pressure fell below 90 in our cases, notwithstanding repeated transfusions, we felt that surgical intervention was indicated. Atropine was not used. With regard to surgery, it seemed that patients below 50 years of age had a better chance to survive with medical measures. Statistically speaking there was not much difference in the mortality rate in patients past 50 years of age whether medical or surgical measures were employed. It must also be borne in mind that bleeding episodes do occur after surgery. In this series the rise in the mortality rate was most striking after 50 years of age.

DR. ALBERT F. R. ANDRESEN, Brooklyn: Care must be taken not to assume that the symptoms which a patient has must be due to an ulcer seen six months or two years before. The symptoms are usually due to something entirely different. The lessons we have learned call attention to the importance of repeated and careful check-up in the presence of any ulcer, large or small, especially if it is in the stomach, and regardless of whether the symptoms have cleared up, because often the persistence of ulcer and of deformity may not be accompanied by intractability of symptoms. There has been pointed out the importance of careful interpretation of x-ray and other findings. I agree with Dr. Jordan with regard to the size of the crater in gastric ulcer as being no indication of whether carcinoma is present or not. We must recognize small carcinomas if we are going to cure people with them at all. Dr. Eusterman showed us carcinomas in young people. All lesions must be frequently checked by x-ray examination to make sure they are not malignant. Large craters may decrease in size as the result of subsidence of edema and inflammation, but irregularities usually remain. The x-ray examination may be a better criterion of carcinoma than the operation. Most cases of duodenal ulcer seen on the operating table are of the complicated type, with large masses of adhesions, induration and old perforation. That is found much more frequently than formerly. Formerly they were not recognized in advance but with more careful spot-film check-up they are seen almost as often as similar gastric ulcer complications and differentiation must be made between irritability and fixation, which can be done by fluoroscopy and the spot films, and persistence of crater as indicating a pocket can also be recognized. As Dr. Portis said, these cases should not be tampered with by medical treatment. They require operation. We should all realize that we should not say that there is a failure of medical treatment in these cases but recognize from the start the surgical indication and follow the patients for a time, with further check-up to make sure we were right that they were surgical from the start.

DR. GEORGE B. EUSTERMAN, Rochester, Minn.: Apropos of some features mentioned by the discussers, I noted in the paper that over one fourth of the carcinomatous gastric lesions of all types give rise to symptoms indistinguishable from chronic benign ulcer and unfortunately may also temporarily respond favorably to treatment. Since the publication of the section program in THE JOURNAL, several physicians have communicated with me and have related experiences similar to my own but have not reported them. It is not the purpose of this paper to serve as a green light to every surgeon to operate indiscriminately on every patient with a gastric ulcer, for we know that the operative mortality and morbidity by and large would be prohibitive. It is also to be remembered that the great majority of such small ulcerous lesions are benign, and many will respond favorably to adequate medical therapy. Case 3 is illustrative of the slow progress of some carcinomas. Symptoms may be present for four or five years. As the muscularis in this instance was intact, the pathologists inferred that the lesion was malignant from the outset. Such long duration of symptoms, taken into consideration with the occasional favorable response of ulcerating carcinomas to treatment, puts the proponents of the carcinomatous transformation of benign ulcer on the defensive. If cases such as I have reported herewith are not periodically checked following treatment, one could easily draw the false conclusion of regarding them as examples of carcinoma on ulcer, or carcinoma developing independently of the ulcer, when the patients returned later with extensive, often inoperable, carcinomatous involvement of the organ.

INTESTINAL AND MESENTERIC INJURY DUE TO NONPENETRATING ABDOMINAL TRAUMA

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Our purpose in this communication is to set forth the results of studies of a surgical problem for which treatment up to now has been significantly unsuccessful, as evidenced by a distressingly high mortality rate. These studies regard the little-understood injuries to the intestine and the mesentery by force which does not penetrate the abdominal parietes, and they were occasioned by recent clinical experiences in which the unfortunate outcome, while expected and reasonably explained, yet seemed preventable. During the past year we have observed 3 patients with rupture of the intestine, in 1 of whom the diagnosis was obvious and prompt operative treatment was followed by recovery. Operation on the other 2 patients was delayed until after the full development of generalized peritonitis, with resulting death. With full realization that recovery might have been possible had the diagnosis been made in the first few hours after injury and with a desire to determine the minimum information necessary to establish a tentative diagnosis leading to earlier surgical intervention, we have studied all available case records in the city hospitals of Atlanta and Cincinnati, and in addition we have studied the experiences of others to obtain such data as seem significant.

EXPERIENCE OF OTHERS

Many points of historical interest were uncovered in tracing the recorded information back to the first writings on this subject. The extreme fragility of the

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intestine was known to Aristotle,[1] who said: "A slight blow will cause rupture of the intestine without injury to the skin." In the middle centuries autopsy reports of intestinal injury due to kicks by horses, blows and falls on the abdomen appeared, and it was noted that many times the injury seemed trivial or insignificant. The first successful operation for subparietal rupture of the intestine is said to have been reported by Sacherus[2] in 1720. Rambdohr[3] in 1730 sutured a complete division of the intestine, with recovery of the patient, and the intact specimen was obtained later when the patient died with pleurisy. In the early part of the nineteenth century experiments were carried out on dogs by St. Croix,[4] Travers[5] and Gross[6] in an attempt to find out the mode of production of intestinal injury and to discover a satisfactory method of suture. In the United States the first case report, by Annan,[7] appeared in 1837, and in 1876 Gregory,[8] in St. Louis, undertook to operate on a patient with such an injury, but the patient died.

At about this time many outstanding surgeons (Sims,[9] Parkes,[10] MacCormac,[11] Lonquet,[12] Curtis[13] and

Appearance of intestine at operation. There is complete transverse division of the jejunum.

Makins[14]) made fervent pleas for surgical intervention in cases of abdominal trauma accompanied with suspected intestinal injury. Many operative attempts resulted, including the operation by Bouilly,[15] whose patient survived for ten days and died after manipulation of the resulting fecal fistula. J. Croft[16] in 1887 operated on a patient with rupture of the ileum and was successful at the first operation, but the patient died four weeks later after attempts to close a fecal fistula.

TABLE 1.—*Comparison of Mortality Rates in Other Series*

Author	Number of Patients	Number Operated On	Number Who Died	Operative Mortality, Per Centage	Number Who Died Without Operation	Total Mortality, Per centage
Royster	1	1	0	0	0	0
Leighton	1	1	0	0	0	0
Senn	1	1	0	0	0	0
Aird	4	4	1	25	0	25
Estes	19	19	10	52	0	52
Stone	4	4	2	50	0	50
Ulrich and associates	1	1	0	0	0	0
Kuhn	4	3	1	33	1	50
Facon and LeCount	8	Not stated			8	100
MacMillan	2	2	1	50	0	50
Dolan	1	1	0	0	0	0
Totten	25	16	5	31	9	56
Lawson	2	2	1	50	0	50
Rowlands	2	2	0	0	0	0
Cope	44	30	21	70	14	80
Wilensky and Kaufman	41	37	22	59	4	63
Mulley	3	3	0	0	0	0
Totals	163	127	64	50.4	36	61.3
Counseller and MeCormack	1,313	887	539	60.7	426	73.4
Grand total	1,476	1,014	603	59.5	462	72.1

However, two years later (1889) a similar opportunity presented itself to Croft,[17] and this time he was successful, with permanent recovery following suture of a perforation of the small intestine. Five months later (October 1889) Moty,[18] in Paris, achieved the same result in a patient who was kicked by a horse in the umbilical area.

We have reviewed the experiences of other surgeons from 1889 to the present. Counseller and McCormack,[19] while adding a case report of jejunal rupture in a child of 8 years, had summarized and tabulated the available case reports up to 1935. They had found records of 1,313 cases, in 887 of which operation was performed and the gross mortality was 60.7 per cent. Diagnosis was established at autopsy in 426, raising the

TABLE 2.—*Age, Sex and Race*

	Number of Cases (36)	Deaths
Age		
20 years or less	14	10
21 to 40 years	12	5
Over 40 years	10	7
Sex		
Male	31	18
Female	5	4
Color		
White	31	18
Negro	5	4

total mortality to 73.4 per cent. Since 1935 163 case reports have been added (table 1), with a decrease in the mortality to 50.4 per cent in cases in which operation has been performed and to 61.3 per cent in all cases.

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3. Rambdohr, cited by MacCormac.[11]
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5. Travers, Benjamin, cited by MacCormac.[11]
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8. Gregory, cited by MacCormac.[11]
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10. Parkes, C. T.: Address in Surgery, M. News **44**: 563 (May 17) 1884.
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ETIOLOGY

Mechanism of Rupture.—We believe there are three chief mechanisms by which nonpenetrating trauma of the abdomen may produce intestinal rupture. Most frequently such perforation results from a crushing injury in which the external force compresses the bowel against the spine or the pelvic bones. Occasionally tearing injuries may result from a violent force applied at a tangent to the body. Bursting injuries of the normal bowel are rare, except those which result from the introduction of compressed air into the anal canal.

In our series of 36 cases the accidents responsible for the injuries were divided as follows: (1) blows on the abdomen by blunt objects (14 cases); (2) falls (5 cases); (3) automobile wrecks (11 cases); (4) kicks in the abdomen (4 cases), and (5) other injuries (2 cases).

Predisposing Causes.—Aird [20] and Wilensky and Kaufman [21] stressed the importance of hernia as a predisposing cause in their series of cases of traumatic rupture of the intestine, and we have found that hernia played an important part in 2 of our cases.

The analysis of our cases failed to show any influence of age on the incidence of intestinal rupture; however, there was a predominance of male patients and of white patients in our series (table 2).

TABLE 3.—*Site of Perforation*

	Number of Cases	Deaths
Jejunum	11	6
Ileum	13	8
Colon	2	2
Appendix	2	0
Mesentery (only)	1	0
Stomach	4	4
Multiple sites	4	3
Bowel, complete severance	6	5
Bowel, massive gangrene	1	1
Duodenum	2	1
Total	36	22

SITES OF RUPTURE

The most frequent site of rupture in our series was the ileum (table 3). There were 4 patients with multiple perforations, 3 of whom died, and 6 patients with complete severance of the intestine, 5 of whom died.

CLASSIFICATION OF CASES

We found it most convenient to classify our cases into three groups. We intentionally excluded cases in which either recovery or death occurred without operation or autopsy. The recoveries were probably few and the deaths many. The three groups of cases are as follows:

Group A.—In this group the diagnosis and treatment were delayed more than twelve hours after the accident. The delay may be attributed to failure of the patient to seek early medical attention because he thought the injury was trivial. On the other hand the delay may have been due directly to the physician's failure to appreciate the seriousness of the injury. In this group, with 17 cases, the mortality was 70 per cent.

Group B.—Diagnosis and treatment in the 14 cases in this group was promptly carried out. Accordingly, the mortality was only 35 per cent.

Group C.—In this group we placed cases of multiple severe injuries which occasionally overshadowed the serious abdominal condition. The mortality from injuries of this kind will always be high, even though treatment is instituted without delay. Our mortality in 5 cases was 100 per cent.

TABLE 4.—*Recovery and Death in Three Groups*

	Recovery With Operation	Death With Operation	Death Without Operation
Group A Treatment delayed more than 12 hours	5	10	2
Group B Treatment prompt	9	5	0
Group C Multiple serious injuries	0	2	3

SIGNS AND SYMPTOMS

Injury due to nonpenetrating abdominal trauma is accompanied by symptoms which vary to such an extent that no clear clinical picture can be set down (table 5). The reason is obvious when one remembers that in the first few hours after injury no sign or symptom may appear. In a large percentage of all cases studied the first symptoms to be noted were those of a complication of the injury, viz. peritonitis.

Further studies of intestinal injuries have produced several mechanical explanations for the delayed appearance of symptoms:

1. Incomplete rupture: In many instances severe bruising of the intestinal wall occurs which sloughs through several hours or even days later because of the development of local necrosis. Spillage is facilitated by the onset of peristalsis after the intake of food or fluids.

2. The production of intestinal paresis by the injury, which inhibits peristalsis for a sufficient time to allow an exudate of plastic lymph to seal the opening. After the patient has recovered from primary shock or after the intake of food, peristaltic action is resumed, with consequent leakage. Cope [22] first described this interesting theory in 1914.

3. Prevention of leakage: Leakage may be prevented mechanically by the plugging of the opening by the mucosal layers or, in the case of complete transverse

TABLE 5.—*Signs and Symptoms*

	Number of Cases (36)	Deaths
Abdominal pain and tenderness	33	19
Nausea	14	10
Vomiting	17	12
Rigidity	28	15
No hepatic dulness	4	4
Dulness in flanks	4	3
Shock	10	7
Coma	3	2
No external abdominal wound	20	13

division, by contraction of the circular musculature of the divided ends.

In a majority of cases shock was not noted when the injured person was first seen but developed as symptoms of peritonitis appeared. In several instances the evidence of shock came on rapidly after a silent period of approximately six hours, which corresponds to the time at which leakage of the intestinal contents into the

20. Aird, Ian: The Association of Inguinal Hernia with Traumatic Perforation of the Intestine, Brit. J. Surg. **24**: 529 (Jan.) 1937.
21. Wilensky, A. O., and Kaufman, P. A.: Subparietal Rupture of the Intestine Due to Muscular Effort, Ann. Surg. **106**: 373 (Sept.) 1937.
22. Cope, V. Z.: Diagnosis and Treatment of Ruptured Intestine, Proc. Roy. Soc. Med. (Sect. Surg.) **7**: 86, 1914.

peritoneal cavity takes place. In other cases (group C) shock was severe when the patient was admitted, principally on account of other severe injuries.

The most constant symptom noted in a high percentage of all reported cases was abdominal pain or tenderness. It frequently is insignificant in the first hours but tends to increase steadily in severity. Tenderness may be localized to the site of the injury but may be elicited only by deep palpation. Cope [22] has stressed the diagnostic value, in rectal examination, of tenderness due to the gravitation of irritating intestinal contents against the pelvic portion of the peritoneum. Vomiting, often associated with nausea, was noted to be the next most reliable symptom.

The pulse rate is the next important indicator. This may be normal when the patient is first seen but tends to rise steadily.

Distention, diminution or absence of hepatic dulness, the appearance of fluid in the flanks and roentgen evidence of air under the diaphragm all are considered positive evidence of peritonitis and are indications for immediate operation. Restlessness, rapid and superficial respiratory movements and definite pain on forced deep breathing are also positive evidence of complications such as hemorrhage or peritonitis.

In our series the only constant signs and symptoms were abdominal pain, tenderness and muscular rigidity. Nausea and vomiting occurred in less than half of the cases. Evidence of free fluid or gas within the peritoneal cavity was rare. The incidence of complete vasomotor collapse was infrequent. It is important, however, to reemphasize the early signs of vasomotor imbalance, such as pallor, diaphoresis, early tachycardia and abnormal blood pressure. These signs are a common feature immediately after accidents. The danger of waiting for signs of peritonitis, gross intestinal hemorrhage or severe shock cannot be overemphasized. External evidence of abdominal injury was present in only 16 of our 36 cases.

METHOD OF TREATMENT

The decision as to when to operate on patients with injuries of the type described requires keen clinical judgment. The importance of instituting treatment promptly is self evident when one compares the mortality in group A with that in group B. We believe it is sometimes wiser to risk an unnecessary abdominal operation (the mortality is low) while the patient's condition is still good than to delay operation, since in cases of perforation there is always a great increase in the mortality. However, since intraperitoneal and extraperitoneal hemorrhage without intestinal perforation may simulate rupture of a hollow viscus, the decision as to when to operate is exceedingly difficult in some cases.

The preoperative treatment for shock and dehydration is of paramount importance. When possible the choice of the anesthetic agent should rest with the specially trained anesthesiologist.

The operative procedure should be thorough but nonshocking. One must guard against overoperating. The choice of a method of intestinal repair varies according to the number and size of the lesions, the viability of the intestine and the general condition of the patient. Simple closure of the perforation, resection of the bowel and even exteriorization of the bowel in cases of severe [illegible] are the most common procedures. Sometimes [illegible]section is indicated and time is a factor the Murphy button may be used to advantage. The question of cleansing the peritoneum is frequently raised. It is our opinion that the gross foreign bodies should be picked off carefully and the contaminated loops of bowel should be cleansed locally with physiologic solution of sodium chloride. This may be done by removing the affected loops from the abdomen and then rinsing them or occasionally by irrigating the loops intra-abdominally in the natural basins which may be formed by rolling or tilting the patient. Irrigation of the entire peritoneal cavity should be avoided. In cases of gross contamination in which treatment is instituted late the local implantation of sulfanilamide or a derivative within the peritoneal cavity and in the abdominal wound may prove a useful adjunct to therapy. This implantation is not advocated as a routine procedure until more convincing experimental work has been done. Closure of the abdominal wall should preferably be done by an expeditious but safe method, such as the use of through and through silver wire or silkworm gut sutures. Drainage of the general peritoneal cavity should not be attempted because of the attendant dangers and inadequacy.

Postoperative care should include adequate intestinal decompression. Proper electrolyte, protein and fluid balances are to be regulated by a careful recording of the fluid intake and output, hematocrit and plasma protein determinations and frequent determinations of the blood chloride levels. Sulfanilamide, sulfathiazole or sulfapyridine administered orally or parenterally is of distinct value in the postoperative care.

SUMMARY AND CONCLUSIONS

The problem of subparietal intestinal injury is concerned with early diagnosis, which in turn calls for prompt treatment before the development of peritonitis. Even under the most favorable circumstances (as in group B) the mortality is 35 per cent, which must be considered high in comparison with that for other surgical procedures, but this figure is doubled (becoming 70 per cent) when operation is performed after the onset of complications (as in group A).

Study of the cause of injury in 1,476 cases revealed the interesting fact that a high percentage of injuries were produced by a relatively small variety of accidents. These included injuries received in personal encounters in which the patient was struck or kicked or fell on the abdomen. Distention of the intestine with food or fluid contributed to the likelihood of rupture, as was evidenced by the fact that many injuries were received soon after the patient had eaten. In industrial accidents, another type, the damage was produced by a blunt object, such as an iron bar or a piece of timber, striking the abdomen with considerable force. The third type of accident by which intestinal injuries were produced was concerned with transportation, the accidents involving the automobile, motorcycle, train, street car, airplane and bicycle, frequently moving at a fast rate of speed. In accidents of this type many forces were applied simultaneously, in that the patient was in motion in one direction and came forcibly against a fixed object or an object moving in the opposite direction, so that a severe crushing injury was produced.

Since it is unquestionably true that intestinal rupture may take place beneath an intact and unbruised abdominal wall and that in some cases no symptoms can be detected during the early hours after injury, if the present high mortality is to be lowered, earlier

surgical intervention is required, and the decision to operate must be based in many instances on intelligent suspicion alone. This suspicion should necessarily be built on a careful study of the accident, with particular emphasis on the exact details of how the abdomen was struck. If the type of accident elicits suspicion, the patient should be observed for four to six hours, and if abdominal pain or tenderness on pressure can be evoked the indications for operation are adequate. A constant rise in the pulse rate makes such a procedure imperative. Decision not to operate is many times more dangerous than the hazards of an unnecessary exploration.

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ABSTRACT OF DISCUSSION

DR. W. L. ESTES JR., Bethlehem, Pa.: In reviewing 2,217 consecutive cases of injuries of all types in our clinic we found that 32 represented abdominal trauma, an incidence of 1.4 per cent. Cody has reported deaths from trauma in Iowa in a three year period as being 5,744 with 53 abdominal traumatic cases, an incidence of 0.9 per cent. Clinically, abdominal trauma may be divided into (1) severe multiple injuries which are rapidly fatal but for which no treatment is of avail, (2) cases that obviously require immediate operation and (3) cases in which the diagnosis is doubtful and the indications for or against operation are not clear. In the proper care of this third group lies the greatest opportunity for saving lives and lowering the extraordinarily high mortality rate. All cases of abdominal trauma, no matter how trivial, in which a decision for operation cannot be immediately made should be kept under the closest observation. There are many cases of abdominal injury on record in which the symptoms of onset were apparently trivial but later, within four or five hours, definite evidence of a severe abdominal lesion, requiring operation, developed. With delay in operation, frequent reexamination should be made at least every thirty minutes—until definite conclusion for or against operation can be reached. Repeat blood counts should be taken every thirty minutes, and no opiate should be given. The upper jejunum and the terminal ileum are the most common locations for subcutaneous rupture or laceration of the intestine, because in these locations the mesentery is short and compression against the vertebral column can occur. Persistent or recurring pain and persistent vomiting with increasing pulse rate are most important. Boardlike, general or localized rigidity and symptoms of a spreading peritonitis will be present in the outspoken case. In the doubtful case an abdominal roentgenogram to demonstrate subdiaphragmatic free air may be helpful but, if negative, does not eliminate the possibility of intestinal rupture. Successful treatment depends entirely on the magnitude of the injury, early diagnosis of rupture, prompt decision that an operation is necessary and the correct time for and method of operative intervention. Decision for or against operation should be made within four or six hours of the accident. It is only by prompt recognition and early operation that the surprisingly high mortality in this deceptive lesion can be effectively reduced.

Unnecessary Investigations.—I admit that there are many occasions, many diseases, many patients who demand any or all of those investigations. What I object to, and what many patients object to, is that they are demanded every time, and they cost money. Indeed, this constant demand for widespread and often unnecessary investigations provides a large part of the reason for the familiar plaint that modern medicine is so expensive. Of course it is expensive if the patient has to run the gamut of all the specialties before he can be told he has a cold in the nose. Judicious selection of the investigations necessary to arrive at a diagnosis is as much a part of the physician's duty as a judicious selection of drugs in treatment.—Atkinson, Miles: Behind the Mask of Medicine, New York, Charles Scribner's Sons, 1941.

THE SIGNIFICANCE OF NECROTIZING PYELONEPHRITIS IN DIABETES MELLITUS

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AND
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While the physician treating patients with diabetes mellitus devotes much attention to the early discovery of infections, the importance of the infections involving the urinary tract has been underestimated. It is our purpose in this paper to discuss a severe form of urinary tract infection in patients with diabetes and the stages by which the final changes come about. The end result is a necrotizing pyelonephritis with extensive destruction of renal tissue. This complication of diabetes, unless recognized in its incipience, leads to a fatal termination. Most of the features of this form of pyelonephritis are seen occasionally in patients not suffering from diabetes. However, the severity, rapid progression and extent of necrosis of renal tissue are hardly to be found in the absence of diabetes.

In some recent surveys of the complications of diabetes, the infections of the urinary tract have not appeared especially common.[1] However, in others,[2] urinary infection has been regarded as a frequent and serious complication of diabetes. The urologist usually sees patients with diabetes after infection is already advanced and often only when irreversible impairment of renal function has occurred. It appears desirable, therefore, to emphasize the incidence of urinary tract infections in patients with diabetes and especially to investigate the early stages of such infections.

Ophüls'[3] studies indicate that 4 per cent of persons coming to necropsy have an infection of the upper urinary tract which may or may not have been the cause of death. It has been demonstrated that about 20 per cent of people dying of diabetes mellitus have infection of the kidneys.[2] Thus, renal infection is found at necropsy five times as frequently in patients with diabetes as in the population at large.[2] However, a much smaller percentage of patients with diabetes are found to have urinary infection during life.[4] It is reported that 6 per cent of 4,500 patients with diabetes mellitus had urinary infection and only 4 per cent needed urologic investigation.[5] The discrepancy between 20 per cent at necropsy [2] and 6 per cent in life [5] is too great to be accounted for by terminal infection alone. The lack of symptoms in the earlier stages and the consequent difficulty in recognizing the infection probably accounts in part for the discrepancy. It seems that a further search for bacteria in the urine of patients without symptoms of urinary tract disease would lead to finding the earlier stages more frequently and hence to earlier therapy.

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From the Departments of Surgery and Pathology of the Harvard Medical School and the Urologic and Pathologic Services of the Peter Bent Brigham Hospital.

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3. Ophüls, William: A Statistical Survey of Three Thousand Autopsies: From the Department of Pathology of the Stanford University Medical School, Stanford Univ. Publ., Univ. Series, M. Sc. **1**: 127-370, (Nov.) 1926.
4. Wilder.[4] Pool.[5]
5. Pool, T. L.: Urological Diseases in Patients with Diabetes Mellitus, Thesis, Minnesota University Graduate School, 1939, quoted by Wilder.[4]

In order to determine the incidence of asymptomatic bacteriuria in ambulatory patients, a group of 50 unselected diabetic patients was compared with another group of patients of similar age distribution in whom diabetes was not present. Although the group investigated is small, the results of the study are so definite that the inclusion of these data seems warranted. While the numbers were small, care was taken to have different age groups equally represented in the two series. Furthermore, the findings have been recorded as negative unless the bacteria or white blood cells were abundant. All female patients were catheterized; the specimens from male patients were voided after careful cleansing of the glans. The presence of bacteria was determined in unstained smears of the urinary sediment after centrifuging. The results were confirmed and the gross classification of the organisms established in smears stained by the method of Gram. The results are given in table 1. It will be noted that bacteria were present in the urine of 27 of the diabetic patients while

TABLE 1.—*Bacteria in the Urine*

	50 Diabetic Patients			50 Nondiabetic Patients		
Decades	No. in Decade	Bacilluria	Pyuria *	No. in Decade	Bacilluria	Pyuria *
Second	3	0	0	3	0	0
Third	2	2	0	2	0	0
Fourth	2	2	0	3	0	0
Fifth	10	5	1	10	0	0
Sixth	14	9	4	12	2	1
Seventh	15	7	3	15	1	0
Eighth	4	2	2	5	1	1
	50	27	10	50	4	2

* There were no cases of amicrobic pyuria. The patients with pyuria are also listed in the heading of bacilluria.

TABLE 2.—*Presence of Bacteria in Urine of Patients with Diabetes as Disclosed by Culture*

Staphylococcus albus	12
Staphylococcus aureus	14
Enterococcus	4
Escherichia coli	8
B. alkaligenes fecalis	1
Staphylococcus albus and Enterococcus	4
Escherichia coli and Enterococcus	3
No growth	4

in the nondiabetic group bacteria were demonstrable in only 4.

Though the significance of bacteria as the only abnormality in the urine may well be questioned, the added presence of pyuria means that invasion of tissues by pathogenic organisms has taken place. The fact that pyuria was found in 10 of the diabetic patients but in only 2 of the nondiabetic group thus becomes of special significance.

Numerous bacteria were present in the urine six or seven times as frequently when diabetes was present as when it was absent. Pyuria was found five times as often among the patients with diabetes as among the controls.

Cultures were made from the urines of patients with diabetes but not from those of the control group. The results are given in table 2. The presence of bacteria in the urine was detected more often by culture than by examining the urinary sediment directly. This discrepancy is accounted for by the fact that the cultures are positive when organisms are present, regardless of their numbers; we judged the sediment negative when studied directly unless bacteria were found in abundance. The organisms found by culture include most of the common invaders of the urinary tract. The incidence of staphylococci is greater than would have been expected from studies in the literature.[6]

In the well controlled diabetic patient, these urinary infections are usually asymptomatic. Their significance lies in the part they play when the patient's resistance is lowered by adverse circumstances. The peculiar fulminating form of necrotizing pyelonephritis to be described may well arise from these mild asymptomatic and often neglected instances of urinary infection in patients with diabetes.

The severe renal infections become manifest clinically by the appearance of fever, pain, hematuria and pyuria. Necrosis of the renal papillae is demonstrable on further investigation. Günther,[7] in describing necrosis of the renal papillae in 10 patients, pointed out that diabetes was present in 7 of them. This stage has been demonstrated by pyelography.[8] Necrosis of the pyramids is by no means confined to pyelonephritis complicating diabetes. As Mallory, Crane and Edwards[9] have pointed out, involvement of the renal pyramids is one of the early lesions of experimental pyelonephritis. Furthermore, the papilla is the most frequent site for the origin of "primary" renal calculi.[10] The irregular filling defect produced in the pyelogram by necrosis of the renal papilla in pyelonephritis bears a striking resemblance to that produced in renal tuberculosis, as shown in the accompanying illustration.

We present in detail the clinical and pathologic findings in 3 patients with progressively more severe necrotizing pyelonephritis complicating diabetes. Case 1 showed changes in the pyelogram early in the disease very much like those found in renal tuberculosis but due to necrosis of the renal papilla as the result of infection with pyogenic organisms. Case 2 presented a more extensive necrotizing infection which terminated fatally. In case 3, study of the kidneys revealed almost complete necrosis of renal tissue. The 3 cases indicate some of the stages through which low grade renal infection manifested only by bacteriuria passes to almost complete necrosis of the kidney.

CASE 1.—Miss V. M., aged 24 years, entered the hospital because of weakness and vomiting for several days. She had received treatment for severe diabetes mellitus over a period of eleven years. One year before admission she was treated for diabetic neuritis and six months before began to experience macroscopic hematuria. A month before, urinary infection due to Escherichia coli was discovered and treated unsuccessfully with mandelic acid and sulfanilamide.

The patient was found emaciated, with a temperature of 103 F. and a dry, red tongue. The blood pressure measured 120 mm. of mercury systolic and 60 mm. diastolic. Nothing abnormal was found on examination of the chest. There was diffuse abdominal tenderness but none in the flanks, and neither kidney was palpable. There was hyperesthesia over the left thigh and on the soles of both feet. Ankle jerks were absent, and the patellar reflexes were hypoactive. The Wassermann and Hinton tests were negative. Urinalysis showed a specific gravity of 1.022, a slight trace of albumin, sugar 4 plus,

6. Braasch, W. F.: Clinical Data Concerning Chronic Pyelonephritis, J. Urol. **39**: 1-33 (Jan.) 1938.

7. Günther, G. W.: Die Papillennekrosen der Niere bei Diabetes, München. med. Wchnschr. **84**: 1695-1699 (Oct.) 1937.

8. Olsson, Olle: Spontanes Gaspyelogramm, Acta radiol. **20**: 578-584 (Dec.) 1939. Günther.[7]

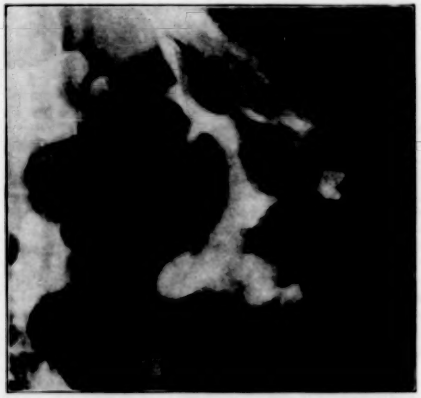
9. Mallory, G. K.; Crane, A. R., and Edwards, J. E.: Pathology of Acute and Healed Experimental Pyelonephritis, Arch. Path. **30**: 330-347 (July) 1940.

10. Randall, Alexander: Papillary Pathology as a Precursor of Primary Renal Calculus, J. Urol. **44**: 580-589 (Nov.) 1940.

tone 1 plus. There were many leukocytes and occasional erythrocytes in each high power field. The hemoglobin of the blood measured 82 per cent (Sahli), the erythrocyte count was 4,260,000 per cubic millimeter, the leukocyte count was 12,800 per cubic millimeter and the smear showed 90 per cent polymorphonuclear leukocytes and 10 per cent lymphocytes. Culture of the urine from the bladder yielded a growth of E. coli. The blood urea nitrogen was 14 mg. per hundred cubic centimeters.

Cystoscopy showed a chronic vesical inflammation without ulceration. The trigon had a granular edematous appearance and the bladder was atonic. The urine from the left kidney contained large numbers of leukocytes on several examinations, while that from the right kidney contained none at any time. Phenolsulfonphthalein excretion appeared in five minutes from the right kidney and in nine minutes from the left kidney. The total excretion in ten minutes from the right was 11 per cent and from the left 1 per cent. Cultures from the left kidney yielded a profuse growth of E. coli and from the right were negative.

The diabetes was controlled by diet and insulin. However, the patient continued to have a fever in the evening which varied from 100 to 103 F. There was no demonstrable response to



A left retrograde pyelogram in case 1, showing irregularity of a middle calix and to a less extent of a superior calix. This is the result of necrosis in the renal papillae. The changes shown in the pyelogram bear considerable resemblance to those produced by early renal tuberculosis.

either sulfanilamide or sulfathiazole. Pyelograms showed distortion of the left renal pelvis, as shown in the illustration.

Left nephrectomy was performed under ether anesthesia. On the third postoperative day the temperature rose to 104 F. and the leukocyte count of the blood to 22,000 per cubic millimeter. The blood urea nitrogen rose to 61 mg. per hundred cubic centimeters and the blood sugar to 770 mg. per hundred cubic centimeters on the fourteenth postoperative day. One week later the right kidney was tremendously enlarged and easily palpable yet nontender. The signs were those of a diffuse pyelonephritis with no evidence of either nephric or perinephric abscess. Improvement was slow but, as infection cleared, the diabetic state became controlled and renal function improved. The patient was discharged from the hospital three months after operation still showing evidence of a chronic urinary infection but the nonprotein nitrogen of the blood had returned to normal. Six months later the patient was well; she had gained 55 pounds (25 Kg.). However, since urinary infection persisted, the prognosis remained guarded.

The kidney removed at operation measured 12 by 6 by 5 cm. From the reddish brown external surface there projected many yellowish white knoblike elevations which varied from 2 to 5 mm. in diameter and which were raised 1 or 2 mm. above the surrounding parenchyma. The markings of the cut surface were irregular. Small white areas were present in the cortex and outer part of the medulla; these were continuous with the elevations on the exterior. The surface of the pelvis was dull grayish white. No areas of ulceration were encountered.

Examination by microscope showed streaklike areas of inflammation extending through the kidney from pelvis to cortex. In these areas the glomeruli were fibrosed and the tubules somewhat dilated. The interstitial connective tissue was increased in amount and was infiltrated with many lymphocytes and a smaller number of polymorphonuclear leukocytes. A few gram-negative bacilli were found in the sections. The knoblike elevations were composed of areas of scar tissue in which all traces of renal parenchyma had been destroyed. The arterioles about these areas of scarring displayed moderate arteriosclerosis but no organized thrombi were seen. There was an extensive lymphocytic infiltration of the subpelvic tissues as well as an increase in connective tissue in this region.

The urinary infection was well advanced for months before its recognition and treatment. When the patient came to the hospital, chemotherapy was without effect. The lack of response is partly explained by failure of the drug to penetrate tissues whose circulation has been greatly diminished by chronic infection and partly by the fact that the diabetic state could not be satisfactorily controlled in the face of persistent infection. Atony of the bladder with accompanying residual urine played an important role in prolonging infection, particularly after operation. There were other evidences of neurogenic disturbances in addition to the vesical disorder, such as hyperesthesia of the legs and feet and loss of tendon reflexes. The hazard of nephrectomy for unilateral pyelonephritis is demonstrated by the extensive infection of the remaining kidney after operation.

Examination of the kidney after removal showed nodular areas of scarring, representing the end result of necrosis of renal tissue. There was evidence of active infection in other parts of the kidney. The process, then, is regarded as one which is progressing in some places and healing or healed in others.

CASE 2.—MRS. E. B., aged 46, came to the hospital because of intermittent attacks of hematuria, frequency of urination, chills, fever, abdominal pain, nausea and vomiting. Mild urinary symptoms had been present for eight months, and the other symptoms had appeared during the previous two weeks. Seventeen years earlier she had had an attack of acute pyelonephritis during a pregnancy which ended in miscarriage. Eleven pregnancies resulted in nine healthy children, one stillbirth and the miscarriage just mentioned.

Lower abdominal tenderness was the only relevant finding on physical examination. The urine contained large numbers of leukocytes and erythrocytes; there was a slight trace of albumin and 2 plus sugar. Renal excretion of phenolsulfonphthalein was measured at 65 per cent the first hour and 25 per cent the second hour. The nonprotein nitrogen of the blood was 31 mg. per hundred cubic centimeters. The only abnormality demonstrated in intravenous urograms was dilatation and irregularity of the right renal pelvis. At cystoscopic examination the bladder was found to be diffusely inflamed; no other cause for hematuria was discovered. Cultures of the urine yielded a growth of Staphylococcus albus and nonhemolytic streptococcus.

After the patient had been treated for twelve days, improvement of the infection occurred and it was possible to control the diabetes by diet alone. The patient was discharged to the outpatient department, where treatment was continued but pyuria persisted.

Five months later dilation and curettage of the uterus was performed for postmenopausal bleeding. This was followed by a febrile reaction and an acute exacerbation of urinary infection. At cystoscopic examination under evipal anesthesia the

mucous membrane of the spastic bladder was covered with a shaggy gray exudate. Ureteral catheters could be passed only 3 cm. on each side. During the next three days the nonprotein nitrogen of the blood rose to 70 mg. per hundred cubic centimeters and the blood sugar to 350 mg. per hundred cubic centimeters, but sugar and acetone did not appear in the urine. The temperature varied between 102 and 103 F. The output of urine gradually diminished in spite of an intake of 3,000 to 5,000 cc. of fluid a day. Though the urinary output was reduced to 200 cc. a day the specific gravity of the urine did not exceed 1.010. The left kidney was palpable and tender. Incision and drainage of the left perinephric abscess and left nephrotomy were performed under local anesthesia. Death occurred soon after operation.

The anatomic diagnoses were acute and chronic pyelonephritis with necrosis and abscess formation, bilateral; multiple perinephric abscesses, bilateral; nephrostomy, recent; acute and chronic cystitis and ureteritis; peritonitis, generalized; glycogen in liver cell nuclei and in kidney.

The right kidney weighed 240 Gm., the left 360 Gm. Both kidneys were surrounded by fat tissue containing numerous small and large abscesses. After this tissue was removed there were uncovered numerous abscesses, some as large as 1.5 cm. in diameter. Beneath the intrinsic capsule was an accumulation of purulent material resulting from rupture of abscesses near the surface of the parenchyma. The kidney markings were almost completely obliterated, and but little uninvolved parenchyma could be made out. The grayish yellow renal tissue was very soft in consistency. The two kidneys were about equally involved in the process, although localized abscess formation was more conspicuous in the left kidney than in the right.

Microscopically the abscesses in the peripelvic fat were found to be composed of necrotic tissue, polymorphonuclear leukocytes and bacteria in large clumps. The general architecture of the renal parenchyma was very much obscured by numerous abscesses like those in the perinephric tissues. The amount of necrosis of renal tissue was large and the abscesses were almost bordering one on the other. Huge clumps of bacteria were found at the center of the abscesses. In the portions of renal tissue not involved in the necrosis there was intense engorgement of blood vessels. Glycogen deposits were found in the cytoplasm and nuclei of the convoluted tubules. The interstitial tissue was increased in amount and was infiltrated freely with lymphocytes and polymorphonuclear leukocytes. The subpelvic tissues showed a similar inflammatory cellular infiltration. The arteries and arterioles showed minimal sclerotic changes. While a few contained small thrombi, the degree of thrombosis did not appear out of proportion to the extent of the inflammatory reaction.

This patient, when first seen, had mild diabetes mellitus and a urinary infection which had been present for several years. In retrospect it is apparent that the latter condition was never adequately controlled. If treatment with sulfonamide compounds had been available at that time, the progress of the infection might have been arrested. However, as shown by case 1, the administration of these drugs must be started early in the course of the disease. While this is important in all instances of pyelonephritis, it assumes even greater significance when the pyelonephritis is complicated by diabetes. The fatal exacerbation of infection in this case followed dilation and curettage of the uterus under ether anesthesia. At necropsy the necrosis of tissue and abscess formation was very extensive. Even if the patient could have survived the infection, the loss of parenchyma would have been too great for renal function to continue satisfactorily.

CASE 3.—Mrs. E. F., aged 46, was admitted to the hospital because of fever, prostration, pain in the flanks and vomiting. The onset of symptoms began with a chill five days previously. Prior to that time she had appeared well and there were no urinary complaints. Eleven years before, during pregnancy, she was told that she had diabetes and hypertension. No regular treatment of either condition was carried out after delivery.

On arrival, the patient was stuporous. The temperature was 104 F., the respiratory rate 35 per minute and the pulse rate 110 per minute. The mucous membranes were dry and the tongue was coated. Scattered rales were heard at the bases of both lungs. The heart was not enlarged. The abdomen was distended and there was a large rounded, nontender mass in the right flank which extended from beneath the costal margin almost to the iliac crest. A similar but smaller mass could be felt in the left flank. There were scattered areas of hyperesthesia over both lower extremities. The patellar and achilles reflexes were absent on both sides.

A plain roentgenogram of the abdomen showed the stomach and several loops of small intestine to be distended with gas. Cystoscopic examination was performed the next day. The bladder contained 200 cc. of foul smelling, bloody urine, and thorough irrigation was necessary to clear the medium. The entire interior of the bladder was covered with adherent clotted blood and gray exudate in the irregular areas.

The Wassermann reaction of the blood was negative. The nonprotein nitrogen of the blood measured 95 mg. per hundred cubic centimeters and the total protein 5.8 Gm. per hundred cubic centimeters with albumin 3.1 Gm. per hundred cubic centimeters and globulin 2.7 Gm. per hundred cubic centimeters. The urine contained a trace of albumin, sugar 1 plus and no acetone, but large numbers of leukocytes and erythrocytes. Studies of the blood showed a hemoglobin content of 75 per cent, erythrocyte count 3,600,000 and leukocyte count 7,900.

A diagnosis of bilateral renal infection was made. Right nephrostomy was thought indicated. Under local anesthesia the right renal fossa was exposed and found occupied by a dense mass of infected, necrotic tissue containing many bubbles of gas. The kidney was a tense, discolored, crepitant sac of necrotic tissue which, when opened, allowed the expulsion of foul smelling gas, clotted blood and urine. Drainage was instituted. The patient died four hours after the operation.

The anatomic diagnoses were acute pyelonephritis with multiple abscesses, extensive, bilateral; infarction and gangrene of kidneys, extensive, bilateral; necrosis of adrenals, extensive, bilateral; cystitis emphysematosa; peritonitis, generalized; pneumoperitoneum; acute splenitis.

The fat in each renal fossa was necrotic and reddish black, with a few yellow areas. Adjacent to both kidneys there were large masses of clotted blood. Both adrenals were reduced to a mass of friable, necrotic material; no viable adrenal tissue could be found. The kidneys were markedly increased in size, the right more so than the left. The renal parenchyma was extremely soft, friable and necrotic. It was infiltrated with numerous bubbles of gas, as were the surrounding tissues. Such structures as the cortex and the medulla could not be identified in the reddish black mass of renal tissue. There were no thrombi visible grossly in the larger branches of the renal arteries and veins.

E. coli was obtained in culture from material in the renal fossa taken at operation, and the same organism from the blood of the heart at autopsy.

There was but little renal tissue which even approached normal in morphology. Abscesses, mostly small, were scattered through the parenchyma in large numbers. The abscesses contained huge masses of bacilli morphologically consistent with E. coli. The tissue among the abscesses showed most extensive anemic infarction. Only after search could one find small groups of relatively normal glomeruli and tubules. Even in these regions there was heavy polymorphonuclear leukocytic infiltration. Slight intimal thickening was found in the medium sized and larger arteries. Many vessels near the abscesses contained partially organized thrombi. As these thrombi were traced into larger vessels they showed progressively less and less evidence of organization. In the largest vessels there was a thin layer of unorganized fibrin over the surface of the intima.

This patient's course is fully accounted for by the fulminating pyelonephritis due to E. coli. The urinary infection had probably been present long before the onset of the terminal illness. However, the patient had no symptoms referable to such an infection until five days before she was admitted to the hospital. The rapid progression of the infection was favored by the presence of diabetes mellitus, which had not been treated for years. The possible significance and gravity of urinary infection as a complication of diabetes is emphasized by the course of events in this case.

At necropsy the kidneys showed a most unusual degree of pyelonephritis with multiple abscesses and, in addition, very extensive infarction and gangrene. It is apparent that the regions of infarction resulted from thrombi which had their origin in small vessels about some of the abscesses. From these sites the thrombi were propagated centripetally, thereby leading to infarction of progressively larger areas of renal tissue. The lesion represents a fulminating pyelonephritis of an extent and degree not found elsewhere in our material and in few cases recorded in the literature. The complete necrosis of both adrenal glands resulting from extension of the renal infection probably was an important factor in the rapid course and fatal termination of the disease.

One would have expected, perhaps, that a renal infection with so many unusual features would have been caused by an uncommon organism. Our cultures, however, yielded only E. coli in spite of the fact that they were prepared aerobically, anaerobically and under partial carbon dioxide tension. A few gram-positive bacilli were also seen in direct smears, but they did not grow out in culture. The large masses of bacteria seen in sections of the kidneys were all gram-negative bacilli and hence were entirely compatible with the cultural findings. E. coli has been found in similar cases recorded in the literature.[11] There are two factors to be considered: one, the nature of the organism, the other, the resistance of the tissues invaded. The findings indicate that it was the second which was responsible for the end result in this case.

THE DEVELOPMENT OF NECROTIZING PYELONEPHRITIS

The ease with which infections are set up in the diabetic state would suggest that bacteria in the urinary tract are at least a potential hazard and one to be much more seriously regarded in the presence than in the absence of diabetes. When pyuria is present (as it was in one fifth of our series of unselected patients with diabetes) it may be assumed that a locus of infection has already been set up in the urinary tract and destruction of tissue has begun. These are the initial steps leading to the disastrous renal infections which have been discussed in the three cases just presented.

Once the infection has gained a foothold, its spread is greatly favored by the presence of diabetes. Thus infections which would be walled off in an otherwise healthy individual tend to spread so as to involve progressively greater amounts of tissue. One of the characteristics of the inflammatory reaction to certain organisms, especially the staphylococcus, is the formation of thrombi about the lesion. These thrombi tend to prevent extension of the infection by closing endothelial lined channels. Such thrombi occur in infections, whether or not diabetes is present. In cases 1 and 2 the thrombi seemed to be in proportions commensurate with those expected to accompany that particular degree of infection. However, in case 3 the thrombi were propagated centripetally. The oldest and most completely organized ones were in small vessels about the areas of infection. The fresher and less organized thrombi were in larger vessels. The propagation of the thrombi, then, must be regarded as an important factor in the extreme degree of necrosis seen in the kidneys in case 3. Abscesses are frequent in many types of renal infections, but such extensive necrosis as that found here is comparatively rare. For this reason the diabetic state seems important in contributing to the end result.

THE RECOGNITION OF URINARY TRACT INFECTIONS IN PATIENTS WITH DIABETES

Certain simple measures may be adopted which will help to make an earlier diagnosis of urinary infection in patients with diabetes. Questions should be directed toward uncovering an alteration of urinary habits and instructions given as to the importance of any change encountered. Unfortunately, the patient who has had polyuria for years as a result of uncomplicated diabetes does not see a warning signal in increased frequency of micturition and may only diminish his fluid intake instead of consulting his physician immediately. The delayed appearance of prominent symptoms demands careful interrogation as to mild dysuria, nocturia, vesical discomfort and pain in the flank. In addition, any systemic manifestation which indicates that the diabetic state has become worse suggests a search for infection, and it is to be remembered that involvement of the urinary tract is not uncommon. Examination of the genitalia leads to the early discovery of phimosis, adherent prepuce (4 of 27 men examined), balanitis, pruritus vulvae and cervicitis. All these conditions may be the antecedents of serious infection in the patient with diabetes. Enlargement of the kidney or perinephrium may be discovered by abdominal palpation before any local symptoms have appeared. In addition to ordinary urinalysis, examination of the stained urinary sediment is of particular value in the early recognition of bacteria in the urine. When present, these should be verified by cultures. If bacteria are found on repeated examination, they should be considered as indicating an active stage of infection even in the absence of pyuria in the patients with diabetes. Glycosuria should of course be controlled and if bacteria continue to be present prompt investigation and treatment should be instituted. It is recommended that in addition to a search for foci of infection, a plain roentgenogram of the abdomen be obtained. Intravenous urography and cystoscopic examination are suggested if they seem appropriate for the study of the individual patient.

Pneumaturia[12] is more common in urinary tract infections in patients with diabetes than it is when this complication is absent. The condition is usually manifested by interrupted urination, which makes the patient aware of the passage of gas. E. coli, Bacillus lactis aerogenes, yeasts and Clostridium perfringens are the organisms that have been found. Analysis of the gas has indicated the presence of carbon dioxide, hydrogen, nitrogen and methane. As a result of fermentation of the urine, butyric acid, lactic acid and alcohol have been

11. Schauwecker, Karl: Ueber Nierenvenenthrombose; Ein Fall mit Gasbildung in der Niere bei Pyelonephritis und Diabetes; Ein Fall bei Amyloid, Virchows Arch. f. path. Anat. **274**: 197-214 (Nov. 14) 1929.

12. Riley, Francis, and Bragdon, Floyd: Pneumaturia in Diabetes Mellitus: Report of Cases, J. A. M. A. **108**: 1596-1599 (May 8) 1937.

recovered. The gas may be formed not only in the urine but also in the tissues of the kidney and bladder in severe forms of infection. Large amounts of gas in the renal tissues were seen at operation in case 3. Olsson [7] has also demonstrated the presence of gas in the renal pelvis by roentgenograms. There have been several reports [13] of cystitis emphysematosa in which the vesical wall is the site of multiple blebs filled with gas. This condition was present in case 3.

If beneficial therapy is to be instituted, it is necessary that infection be discovered early. Chemotherapy is well tolerated if the diabetic state can be controlled. Regulation of the diabetes, however, is difficult once infection has gained the upper hand. When a chronic inflammatory process has been established, impairment of circulation (by fibrosis, suppuration and thrombosis) prohibits an effective permeation of the tissues by a chemotherapeutic agent. Early drainage of infection, a proper diet, forced fluids and increased administration of insulin are general measures which apply in the treatment of any type of infection in the patient having diabetes mellitus. We have in sulfathiazole an effective agent for combating staphylococcic infections in general, but its efficacy in the management of urinary tract infections is limited by the factors outlined. Prophylactic use of this drug in small doses for patients with diabetes when bacteria are found in the urine in the absence of symptoms referable to the urinary tract may prevent the development of a serious infection with destruction of renal tissue.

Maintenance of the caloric intake is of particular importance in chronic infections. The insulin requirement is greatly increased in the face of infection and, as Wilder [14] has pointed out, the patient may need insulin then even if he did not need it before. The maintenance of fluid intake at as high a level as possible is of great importance for the patient with diabetes complicated by renal infection and cannot be overemphasized. When surgical intervention is necessary, local anesthesia is much to be preferred to any form of general anesthesia. The introduction of any factor which tends to cause dehydration increases the difficulty of controlling acidosis.

In the treatment of these infections, it is of the utmost importance to bear in mind that destruction of renal tissue must be prevented or stopped at the earliest possible moment. Parenchyma, once destroyed, cannot be replaced. Not only is the loss of renal tissue important in itself but there may also be serious consequences outside the kidney because of the production of vascular lesions in many organs.[15]

CONCLUSIONS

1. Asymptomatic urinary infections are much more frequent in patients with diabetes than in those without this condition.

2. The asymptomatic urinary infections in the diabetic state may lead to serious lesions of the kidneys. Necrosis of renal tissue extends rapidly, once the infection gains a foothold. It is increased in some cases by centripetal propagation of thrombi which form about the areas of infection.

3. A consideration of the end results of urinary tract infection in patients with diabetes emphasizes the importance of treating the infections before irreplaceable tissues have been destroyed.

101 Bay State Road.

13. Mills, Ralph G.: Cystitis Emphysematosa, J. Urol. **24**: 217-231 (Sept.) 1930. Levin, H. A.: Gas Cysts of the Urinary Bladder, ibid. **39**: 45-52 (Jan.) 1938.

14. Wilder, Russell M.: Clinical Diabetes Mellitus and Hyperinsulinism, Philadelphia and London, W. B. Saunders & Co., 1941.

15. Weiss, Soma, and Parker, Frederick, Jr.: Pyelonephritis: Its Relation to Vascular Lesions and to Arterial Hypertension, Medicine **18**: 221-315 (Sept.) 1939.

ABSTRACT OF DISCUSSION

DR. THOMAS P. SHUPE, Cleveland: The clinician as well as the pathologist does not know much about the subject. That it occurs quite frequently in diabetes has been shown by Guenther, whom the authors mentioned and that was brought out in 1937. The authors cited 1 case of a man aged 60 with no sugar in the urine and ascending urinary infection. He had a hydronephrosis and blockage by a large stone in the ureter. The kidney removed showed extensive papillary necrosis. On investigation the blood sugar showed an elevation of 170 to 200. Later, death and autopsy revealed the same necrosis on the other side. The difficulty with these cases has been that the diagnosis was made in reverse; that is, from the autopsy table back to the laboratory. The nondiabetic may show papillary necrosis, but nobody has reported such cases except in the advanced cases of carcinoma of the prostate and of the bladder. The pathologist making an autopsy on a patient with a dilated stomach and fatty degeneration of the liver, a yellow cranium and a firm brain should look for the papillary necrosis and other signs of diabetes. There is no doubt that the diabetic patient is subject to many hazards even if he has sugar in the urine. The diabetic patient without sugar in the urine is perhaps subject to a great many more hazards, and, next to the toes, perhaps the genitourinary system is the most vulnerable. The authors have shown in the slides the papillary necrosis which occurs in these cases. It is a question how much diagnostic and operative procedure should be done in these cases. I should think as little as possible. Whether a retrograde pyelogram is justifiable until the diabetes is well under control is another question; certainly no surgical procedures are except for unblocking of obstruction in the genitourinary tract. Another lesion that must be considered in this connection is the cord bladder with degeneration of the posterior columns due to diabetes. Of course, then one is going to have the residual urine with infection and the general debilitation of the patient. From the rapidly spreading infection which exists in these cases, I think it would be well in all cases of genitourinary infection and obstruction to investigate the level of the blood sugar as well as the blood urea, the nitrogen and the creatinine.

DR. ORVILLE T. BAILEY, Boston: In the general behavior of any infection there are two factors to be considered: One of these is the character of the organism concerned; the other is the state and character of the tissues attacked by the organism. In the infections which we have discussed here there is a striking instance of an unusual result from a familiar group of organisms. Escherichia coli, staphylococci and so on have produced extensive spreading necroses. We must search for the explanation of these lesions not in the nature of the organism but in the character of the tissues infected. Certainly the presence of diabetes is important in bringing about this result. Necrosis of the papillae, in spite of the amount of attention devoted to it in the literature, can hardly be regarded as an isolated lesion. It is a part of a necrotizing process which is demonstrable clinically, especially by the retrograde pyelogram, but does not represent the whole of the condition. By the time necrosis of the papilla is demonstrable the infection has already spread far up into the parenchyma. The unusual character of response to Escherichia coli is especially borne out in the third case presented here. The organism produced a massive infiltration of the tissues with gas, not only of the kidneys but of the perirenal tissues, and the necrotizing process led to complete destruction of both adrenal glands. The infection not only resulted in abscesses but in almost complete gangrene of both kidneys. Careful search of the main vessels failed to show any thrombi. On careful investigation of the small vessels there were thrombi beginning peripherally and extending centrally, thus progressively shutting off the blood supply, resulting in a spreading gangrene of the renal parenchyma.

THE MECHANISM OF DELAYED WOUND HEALING IN THE PRESENCE OF HYPOPROTEINEMIA

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During the past decade protein deficiency has been shown to have an important influence on many of the problems of clinical surgery. Gastrointestinal motility, especially gastric emptying after anastomosis of the stomach and jejunum, was shown to be profoundly retarded by protein depletion,[1] and it was shown that in certain instances "vicious circle" can be relieved by transfusion.[2] Goldschmidt, Vars and Ravdin[3] demonstrated that the protein balance of the body profoundly influences the vulnerability of the liver to chloroform. As a corollary to this principle, diets high in protein as well as carbohydrate were recommended in the treatment of patients with extensive hepatic disease.[4] It is believed that such diets will also prove of great value in the preparation of the thyrotoxic patient for operation.

Fig. 1.—Biopsy specimen of wound in dog 511 taken seven days after the original incision. The serum protein concentration was 2.08 Gm. and the acacia level 3.52 Gm. per hundred cubic centimeters. (Reduced from a photomicrograph with a magnification of 300 diameters.)

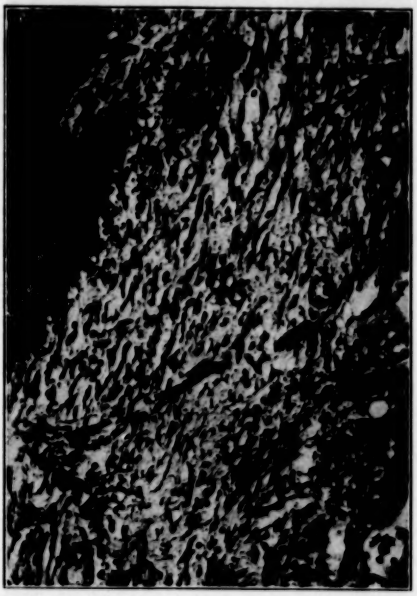

Fig. 2.—Biopsy specimen of wound in dog 42 taken seven days after the original incision. The serum protein concentration was 1.91 Gm. and the acacia concentration 2.38 Gm. per hundred cubic centimeters. (Reduced from a photomicrograph with a magnification of 300 diameters.)

The relation of hypoproteinemia to wound disruption was first demonstrated in the dog by Thompson, Ravdin and Frank[5] in 1938. They showed that the hypoproteinemic dog is frequently incapable of normal fibroplasia. Another systemic factor interfering with wound healing is C avitaminosis. The importance of this factor was clearly demonstrated in the guinea pig by Lanman and Ingalls[6] in 1937.

It has since been a matter of considerable interest to know whether the experimental data are of importance clinically. At the 1940 annual session of the American Medical Association, Hartzell, Winfield and Irvin[7] reported on the serum protein and serum ascor-

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Owing to lack of space, this article has been abbreviated for publication in THE JOURNAL by omission of figure 6. The complete article appears in the authors' reprints.

Read before the joint meeting of the Section on Orthopedic Surgery and the Section on Surgery, General and Abdominal, at the Ninety-Second Annual Session of the American Medical Association, Cleveland, June 6, 1941.

1. Mecray, P. M.; Barden, R. P., and Ravdin, I. S.: Nutritional Edema: Its Effect on the Gastric Emptying Time Before and After Gastric Operations, Surgery **1**: 33 (Jan.) 1937.

2. Barden, R. P.; Ravdin, I. S., and Frazier, W. D.: Hypoproteinemia as a Factor in the Retardation of Gastric Emptying After Operations of the Billroth I or II types, Am. J. Roentgenol. **38**: 196 (July) 1937.

3. Goldschmidt, Samuel; Vars, H. M., and Ravdin, I. S.: The Influence of Foodstuffs on the Susceptibility of the Liver to Injury by Chloroform, and the Probable Mechanism of Their Action. J. Clin. Investigation **18**: 277 (May) 1939. Ravdin, I. S.; Vars, H. M., and Goldschmidt, Samuel: The Nonspecificity of Suspensions of Sodium Xanthine in Protecting the Liver Against Injury by Chloroform, and the Probable Cause of Its Action, ibid. **18**: 633 (Nov.) 1939.

4. Ravdin, I. S.: Some Recent Advances in Surgical Therapeusis, Ann. Surg. **109**: 321 (March) 1939.

5. Thompson, W. D.; Ravdin, I. S., and Frank, I. L.: Effect of Hypoproteinemia on Wound Disruption, Arch. Surg. **36**: 500 (March) 1938.

6. Lanman, T. H., and Ingalls, T. H.: Vitamin C Deficiency and Wound Healing, Ann. Surg. **105**: 616 (April) 1937.

7. Hartzell, J. B.; Winfield, J. M., and Irvin, J. L.: Plasma Vitamin C and Serum Protein Levels in Wound Disruption, J. A. M. A. **116**: 669 (Feb. 22) 1941.

[Footnote 3 continued in next column]

bic acid levels of 20 patients with wound disruption. Hypoproteinemia was present in every patient, and the ascorbic acid level was low in all but 1. C. P. Rhoads [8] has recently stated that in a group of patients

Fig. 3.—Biopsy specimen of wound in dog 43 taken seven days after the original incision. The serum protein concentration was 1.99 Gm. and the acacia concentration 2.77 Gm. per hundred cubic centimeters. (Reduced from a photomicrograph with a magnification of 300 diameters.)

with wound disruption encountered at the Memorial Hospital, New York, hypoproteinemia has been present more frequently than C avitaminosis. We have since had the opportunity of observing 2 patients with wound disruption in whom hypoproteinemia was present with a normal blood ascorbic acid level, and it is our impression that a protein deficit of sufficient magnitude to retard wound healing seriously is more common than a C avitaminosis of sufficient severity to have an equally deleterious effect. Rhoads and Kasinskas [9] more recently have shown that the plasma-depleted dog is slow to form bony callus after experimental fracture.

In the animals studied by Thompson and his associates the hypoproteinemia was induced by repeated plasmapheresis, the animals being maintained on a diet containing less than 1 per cent of protein. This diet was regarded as adequate with respect to all known vitamin and mineral requirements. The serum protein level was reduced gradually over a period of two to four weeks to edema levels (usually under 3.5 Gm. per hundred cubic centimeters). It is known, from the work of Whipple [10] and others, that this procedure depletes the labile protein stores of the body. It results also in a substantial reduction in the plasma volume. Ascites is frequent as well as edema of the extremities. The intraperitoneal tissues are waterlogged, as are the muscles of the abdominal wall. It is evident, therefore, that these dogs suffer in two important regards: (1) They have been for some time in negative nitrogen balance and have a general nutritional deficit of protein, and (2) their colloid osmotic pressure is seriously reduced, with a resultant disturbance in the relation between intravascular and interstitial fluid.

It is obvious that the formation of any new cells, such as fibroblasts, requires protein. Whether this protein would have to be derived from dietary protein or from the stores of labile protein or whether it could be derived from other fixed proteins or the products of their catabolism is uncertain. There is also the possibility that the disturbed osmotic relations in the hypoproteinemic dog might interfere with the utilization of available protein for the production of fibroblasts.

While it was not possible with the means at our disposal to investigate fully the intermediary metabolism of protein, it was possible to restore the osmotic properties of the plasma of the hypoproteinemic dog by the administration of acacia without contributing to the protein intake. Seven animals were prepared in which the protein levels were below 3.5 Gm. per hundred cubic

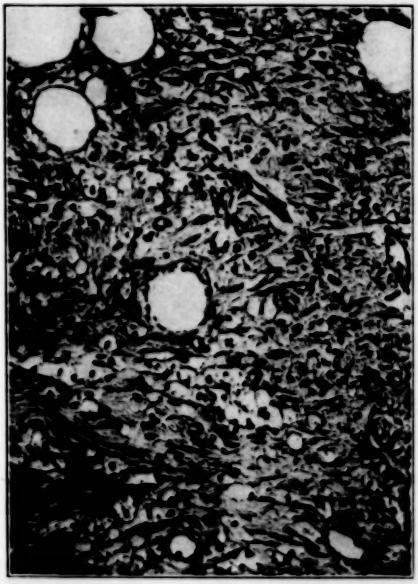

Fig. 4.—Biopsy specimen of same wound as in figure 1 taken at fourteen days. (Reduced from a photomicrograph with a magnification of 300 diameters.)

centimeters but in which there was enough acacia circulating in the plasma to prevent any tendency toward edema. The concentration of acacia was maintained between 2 and 3.6 Gm. per hundred cubic centimeters.

8. Rhoads, C. P.: Personal communication to the authors.
9. Rhoads, J. E., and Kasinskas, W.: Surgery, to be published.
10. Whipple, G. H.: Protein Production and Exchange in the Body Including Hemoglobin, Plasma Protein and Cell Protein, Am. J. M. Sc. 196: 609 (Nov.) 1938.

Three methods were used in the preparation of the dogs. The first 2 were prepared by the repeated administration of acacia solution according to the method of Yuile and Knutti.[11] The observations of these authors that this procedure reduces the fibrinogen concentration and also reduces the serum protein concentration were fully confirmed. Their suggestion that the hemorrhagic tendency observed in some of these dogs might be due to a prothrombin deficiency was not completely substantiated. The prothrombin concentration was at times moderately reduced but not to a point low enough to explain the bleeding tendency without reference to some other factor. Whether there was any other factor, except the low fibrinogen level, we do not know.

Fig. 5.—Biopsy specimen of same wound as in figure 3 taken at fourteen days. (Reduced from a photomicrograph with a magnification of 300 diameters.)

The second method, employed on 2 dogs, was a modification of the procedure for total plasmapheresis described by Stanbury, Warweg and Amberson.[12] While the dog was bled from the femoral artery an infusion of erythrocytes suspended in a solution of acacia was given. The procedure was not carried as far as it had been in the experiments conducted by Stanbury and his associates.

The third method, employed on 3 dogs, consisted first in the preparation of hypoproteinemic dogs by the same technic used by Thompson, Ravdin and Frank.[8] After the hypoproteinemia had been established the test wounds were made and infusions of acacia begun. This procedure had the advantage of depleting the "labile" stores of protein of the animal as well as of reducing the plasma protein concentration. In all 7 dogs incisions were made by aseptic technic through the skin, the subcutaneous tissues, the rectus sheath and the rectus muscle on the right and the left sides. The incisions were approximately 2 inches (5 cm.) long.

Fig. 7.—Scanty fibroplasia in plasma-depleted dog fourteen days after original incision. The incision is at the upper right. (Thompson, Ravdin and Frank.[8])

Fig. 8.—Normal fibroplasia at seven days in a dog whose proteins had been restored by the administration of large amounts of lyophil serum. (Thompson, Ravdin, Rhoads and Frank.[8])

11. Yuile, C. L., and Knutti, R. E.: Blood Plasma Proteins as Influenced by Intravenous Injection of Gum Acacia: Production of Chronic Hypoproteinemia, J. Exper. Med. **70**: 605 (Dec.) 1939.
12. Stanbury, J. B.; Warweg, Edna, and Amberson, W. R.: Total Plasmapheresis, Am. J. Physiol. **117**: 230 (Oct.) 1936.

The rectus sheath was closed on the right side with interrupted sutures of catgut and on the left side with interrupted sutures of silk. On both sides the edges of skin were approximated with interrupted sutures of silk, which were removed on the fifth day. Biopsy specimens were taken at right angles to the wound on the seventh

Fig. 9.—Normal fibroplasia at fourteen days in a dog whose proteins had been restored by the administration of large amounts of lyophil serum. (Thompson, Ravdin, Rhoads and Frank.[13])

and fourteenth days. All the wounds showed definite fibroplasia. Photomicrographs of specimens from the wounds of the last 3 dogs are shown (figs. 1 to 5). In comparison, photomicrographs from the paper of Thompson, Ravdin and Frank [8] disclosing the absence of fibroplasia in hypoproteinemic dogs are shown (figs. 6 and 7), as well as photomicrographs from the paper of Thompson, Ravdin, Rhoads and Frank [13] disclosing normal fibroplasia obtained in dogs whose hypoproteinemia had been corrected by the administration of large amounts of concentrated serum (figs. 8 and 9).

COMMENT

It is evident from these experiments that the formation of fibroblasts is not a function of the concentration of the serum proteins. In fact, in the last 3 animals which received acacia the protein concentration dropped from 3.67 to 1.91, from 3.55 to 1.99 and from 3.11 to 2.08 Gm. per hundred cubic centimeters, respectively, within seven days after the administration of the first infusion of acacia solution. One of the animals had had a decubitus ulcer near the ankle for a week prior to the administration of acacia; during this time it had shown no tendency to heal. Within twenty-four hours after the acacia was administered this ulcer was covered with a dry scab, and healing proceeded rapidly during the next seven days. Another animal had an open sore which had persisted for a week. This also had practically healed within a week after the administration of acacia had been started. The following hypotheses have been suggested in explanation of the effect of the acacia:

1. The relation of hypoproteinemia to wound healing is mainly on an osmotic basis. The acacia corrects the low colloid osmotic pressure of the serum, with the result that fibroplasia occurs.

2. The relation of hypoproteinemia to wound healing is mainly on a nutritional basis, but the administration of acacia makes it possible for more protein to be withdrawn from the plasma for tissue-building purposes.

The liver appears to be full of acacia on histologic examination (fig. 10). The acacia may reduce the affinity of the liver for labile protein and thus make available for tissue repair a larger proportion of the small amount of protein in the diet and perhaps a larger

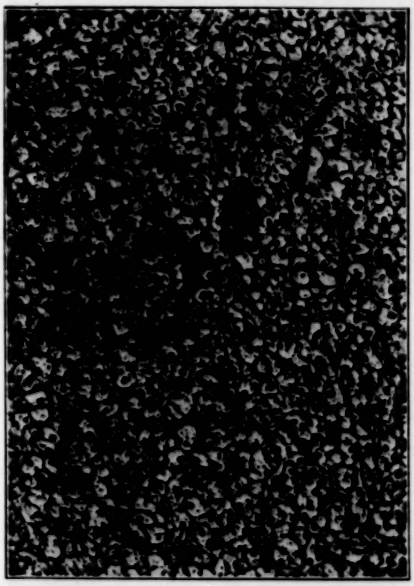

Fig. 10.—Section of liver of dog 511 after administration of 1,500 cc. of 6 per cent acacia solution in a two week period.

proportion of the products of normal protein catabolism. It is not possible at present to decide which of these hypotheses is correct or whether both are important.

CONCLUSIONS

Fibroplasia occurs in the hypoproteinemic animal if ample amounts of acacia are given intravenously to maintain the colloid osmotic pressure of the plasma.

13. Thompson, W. D.; Ravdin, I. S.; Rhoads, J. E., and Frank, I. L.: Use of Lyophile Plasma in Correction of Hypoproteinemia and Prevention of Wound Disruption, Arch. Surg. 36: 509 (March) 1938.

This occurs even if chronic hypoproteinemia is produced so as to deplete the stores of labile proteins. It is evident that the failure of fibroplasia in the hypoproteinemic animal is not directly a function of the concentration of the serum proteins. Acacia solution is not recommended in the treatment of hypoproteinemia in patients.

ABSTRACT OF DISCUSSION

DR. CHARLES G. JOHNSTON, Detroit: Dr. Ravdin and Dr. Rhoads have pointed out the effect of low serum protein on wound healing and the importance of nutrition in surgical patients. Hartzell and his associates have pointed out the relationships of vitamin C to wound healing in patients and in experimental animals. These contributions have made us aware of the importance of nutrition for surgical patients. It is significant that, in the cases of wound disruption which we have studied, the majority have been in patients who have had gastric or duodenal lesions, chiefly ulcer patients, and a majority of them have been on diets. We have recognized definitely, and Dr. Rhoads has intimated, that serum protein determinations do not necessarily mean that we are measuring the ability of the organism to mobilize the protein or to utilize it. More to the point is the fact that plasma vitamin C determinations give little if any information regarding the vitamin C content of the cellular elements. The paper of Dr. Rhoads and his co-workers has afforded proof that the colloidal osmotic pressure is of importance in wound healing. However, I have noted in patients in whom disruption has occurred that the serum protein was only in rare instances low enough to be considered below an edema level. I wish I could explain why an animal with low serum protein might be restored to normal wound healing after the administration of acacia. The data presented do not intimate that in patients who have difficulties with wound healing one should recommend acacia infusions. I prefer Dr. Rhoads's second explanation of the way in which acacia assists, namely the mobilization of protein for utilization. Wound healing is not a simple phenomenon but depends on many factors. It cannot be explained on any one basis, but all factors must be brought into play. As far as nutrition is concerned, serum protein and vitamin C alone are not the entire answer. We cannot ascribe to any one factor exclusive importance in the healing of wounds.

DR. JONATHAN E. RHOADS, Philadelphia: I want to thank Dr. Johnston for emphasizing that acacia is not a suitable agent to use in patients with hypoproteinemia with the idea of improving their wound healing. To maintain concentrations of acacia of 2 or 3 Gm. per hundred cubic centimeters in the blood of these dogs for two weeks, it was necessary to administer a volume of 6 per cent acacia, equivalent to approximately three times the normal plasma volume. You saw what changes were induced in the liver by this amount of acacia, and as long as it is so easy now to get plasma for transfusion it seems no longer justifiable to use acacia in treating human patients.

The Specialty Boards.—There is another criticism of the special boards as at present constituted, which is that they lay too much emphasis on specialization. In an entirely laudable effort to ensure that every doctor who presumes to call himself a specialist shall be entitled by reason of his training to do so, they have made it well nigh impossible for a man to obtain a solid general foundation. This is what happens. The board demands first a year of general work as an intern or in practice, followed by three years of work at the specialty, whichever it may be. This may be done as two years as a hospital resident and one year in practice as assistant to a senior man, or the whole three years may be spent in hospital, though not many hospitals have facilities for a full three years residency. However, this is what is considered the optimum, and these requirements, which in the early days of the boards—and they are still quite young—had to be elastic to allow of the established men taking them, will shortly become absolute.—Atkinson, Miles: Behind the Mask of Medicine, Charles Scribner's Sons, 1941.

SOME MENTAL MECHANISMS IN ALCOHOLISM

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Many approaches and many methods are and have been in use in the study of alcoholism—social, religious and economic as well as scientific. Special societies ranging from the Association for the Study of Alcoholism, with its objective approach, to Alcoholics Anonymous, with its emotional revivalistic urge, are engaged in this problem. Medical investigation likewise has used different technics. Biochemical, physiologic and experimental psychologic research are productive of information. Neuropsychiatry has used various methods from restraint and seclusion to psychoanalysis and attempts at personality education or reeducation in its efforts. This paper deals with a study of alcoholic but otherwise successful persons with whom such attempts were made and some of the mental mechanisms that were found during the course of these attempts.

The source material may be called selected in that it is composed of 105 patients representing 164 of the last 2,000 admissions to a private psychiatric sanatorium. This of itself rules out marginal and indigent economic groups, which differs from Wittman's series.[1] The diagnosis for purposes of classification was "without psychosis, alcoholism." This paper is not as inclusive as the papers of Wall [2] but conforms more to Karpman's [3] remarks in discussion of the former's paper.

Business and professional men made up the greater part of the group and a number of farmers, a few representatives of the arts, a few housewives and career women completed the group. Many were college graduates. The general intelligence level was adult or superior adult. In other words, these persons were considered successful in their sphere whether metropolitan, small town or rural. An early question asked was how they came to be alcoholic. As more contacts were made and more similarity in reaction types was noted, the next problem was how to combat the difficulties that these persons had and how to help them. Out of this experience have come some impressions outlined hereafter.

Most patients entered under the influence of alcohol or with a history of recent overindulgence. An old conception of three stages in management has been followed: During the first stage, while care for the physical features is carried on, some idea of the patient's underlying make-up, freed by alcohol from inhibitions normally present, may be ascertained. During the second stage, when indirect toxic features are still present, when remorse, conscience, apprehension over the effect of disability on business, professional, social or family situation exist, conversations may reveal some conflicts that lead to escape through alcohol. The patient is still shaky emotionally even if not physically. So the trend of conversation runs in that direction with various inferences and sequestrations that may be brought out.

Read before the Section on Nervous and Mental Diseases at the Ninety-Second Annual Session of the American Medical Association, Cleveland, June 4, 1941.

1. Wittman, M. P.: Developmental Characteristics and Personalities of Chronic Alcoholics, J. Abnorm. & Social Psychol. **34**: 361-377 (July) 1939.

2. Wall, James H.: A Study of Alcoholism in Men, Am. J. Psychiat. **92**: 1389-1402 (May) 1936; A Study of Alcoholism in Women, Am. J. Psychiat. **93**: 943-956 (Jan.) 1937.

3. Karpman, B.: Discussion of Wall,[2] Am. J. Psychiat. **93**: 953 (Jan.) 1937.

Persons in this group are sensitive and want at this time sympathy and a justification or rationalization for their drinking, even though they may not ordinarily show it. Recognition of this part of their make-up permits them to unburden. It should be stated here that no attempt to secure transference is made in this stage when emotional factors predominate or in the third stage when the person has resumed the habitual type of response. During the third stage, when the person feels well physically, is outwardly at least settled down and is engaging in activities with associates, more objective conversations based on history and information acquired in earlier observations can be carried on.

TABLE 1.—*Characteristics Studied in One Hundred and Five Alcoholic Patients Without Psychosis*

Economic adjustment	Homoeroticism	Pathologic response to alcohol
Success in occupation	Heteroeroticism	Deterioration
Intellectual level	Filial overdependence	Superficiality
Social attributes	Emancipation	Oral traits
Affective response	Forcefulness	Reaction to masturbation
Adaptability	Perseverance	Inflexibility
Activity	Aggressiveness	Scrupulosity
Extrovert-introvert	Feeling of inferiority	Consistency
Gregariousness	Feeling of guilt	Apprehension
Solitary drinking	Sensitivity	Suspiciousness
Communicativeness	Mood: Cheerful	Preoccupation
Responsibility, general	Depressed	Day dreaming
Responsibility, alcohol	Irritable	Depersonalization
Self control	Hypochondriasis	Alcoholism in family
Attitude to habit	Narcissism	
Sexual adjustment	Anorexia	
Marital adjustment	Sleep disorder	

Forty-eight characteristics combined from several sources [4] were used, as shown in table 1. The information was assembled from histories, self estimates, notes and personal observations. Analysis of these characteristics naturally depends on the approach of the analyzer. An attempt has been made to use as nearly an objective or laboratory approach as possible. No brief is held for any school of psychiatric thought save a study of the total personality. Such a study covers many classifications not mentioned, but even then headings were difficult to find for some outstanding expressions of individual statements. Predominating characteristics are shown in table 2.

It is obvious that the patients in this series were not able to adjust satisfactorily or they would not have come under observation. Nevertheless, as indicated previously, their economic adjustment was such that they or their families could take care of hospitalization; 90 of the 105 could be definitely classed as successful in their occupations; 88 were counted as having satisfactory social attributes; 68 showed good affective response. Adaptability was fairly high in that 63 had a plus rating, while activity was higher than most characteristics with 97 persons marked positively. A corollary to these figures might be found in an attempt at extrovert-introvert typing, with 79 classed in the former, 22 in the latter and 4 in the unclassified grouping. It is recognized that this is an artificial grouping, as it is difficult to classify many persons as pure extrovert or introvert. Gregariousness ran ahead of the extrovert grouping; 90 persons were noted as having that tendency. It is difficult to assay this in the light of the finding that 95 were counted as solitary drinkers. I have taken the position, however, that the person who drinks at home, in his club, hotel or tavern and communes with one person, whether spouse, waiter or barkeeper, is still a solitary drinker in the psychologic sense. The communicativeness which 94 people showed raises the question of whether the attempt of the alcoholic to fraternize does not represent a tendency of the introvert to break through natural inhibitions toward social contact or perhaps social contact with a superior group. One must always consider, in evaluating the foregoing reactions, the effect of alcohol in loosening acquired inhibitions, thus revealing latent active personality traits.

General responsibility ran high, as would be expected in this group, with 80 accepting it. Responsibility as to alcohol, self control with respect to it and attitude toward the habit were low, as would also be expected, with 7, 3 and 25 persons respectively showing anything at all of a constructive reaction. This last category is an important one from the point of therapy. It is obvious that it is only through attempting to develop a positive attitude in the patient that anything is going to be accomplished. The ways and means of developing it through various psychiatric technics are less important than the imperative need for it.

Any arrangement of personality studies must include sexual adjustments in their direct and indirect relationships. Sixty-three were considered as having made sexual adjustment, 30 as not having done so and 12 were questionable. Ninety-three were or had been married. Fifty-two had made a satisfactory marital adjustment, 32 had not, 3 had a plus or minus rating and 6 had a questionable one. Relatively few patients showed overt homosexual tendencies. Not many showed the latent tendencies frequently referred to in alcoholism with expressions in parties, groups and the like. Perverse sexual tendencies were not frequently found. The chaotic sexuality referred to by Rosanoff [5] was evident and, on a percentage basis, even outstanding. Marital maladjustments past and present were frequent. Resort to alcohol in the hope of sexual stimulation for incapacity was rare, though impotence was not infrequent. Most of the marital difficulties arose out of a conflict over the patient's habit. Where they did occur in this group they were mostly social and domestic, which is part of the chaotic sexuality. Conflict on an economic basis over failure to provide occurred only twice.

Filial dependence and/or emancipation therefrom may be considered from the psychoanalytic approach or it may be taken as part of the growing up process.

TABLE 2.—*Characteristics Frequently Found in One Hundred and Five Alcoholic Patients Without Psychosis*

Economic adjustment	96	Sexual adjustment	63
Success in occupation	90	Marital adjustment	52
Intellectual level plus	85	Filial overdependence	43
Social attributes plus	88	Emancipation	68
Affective response	68	Forcefulness	46
Adaptability	63	Perseverance	68
Activity	97	Aggressiveness	52
Extrovert	79	Feeling of inferiority	41
Solitary drinking	95	Anorexia	48
Communicativeness	94	Sleep disorder	81
Responsibility, general	80	Superficiality	90
Gregariousness	90	Alcoholism in family	52

Records of 43 indicated filial overdependence, while 68 showed emancipation from it. Twenty-nine of the dependent ones failed to develop freedom. Undoubtedly there were others as well in whom the overdependence was latent if not manifest. Escape through alcohol to a false feeling of emancipation was shown frequently in resentment toward parents in expressions

4. Bigelow, N. J. T.; Lehrman, S. R., and Palmer, J. N.: Personality in Alcoholic Disorders, Psychiat. Quart. **13**:711-720 (Oct.) 1939. Clark, S. N., Personal communication to the author. Wittman,[1] Wall,[2] first reference.

5. Rosanoff, A. J.: Manual of Psychiatry and Mental Hygiene, ed. 7, New York, John Wiley and Sons, 1938, p. 374.

such as drinking being one way of showing independence. While outwardly conforming at most times, the inability to be normally independent led them to desire it abnormally—hence alcohol.

Forcefulness was not an outstanding characteristic. Less than half received such a rating. Genuinely forceful persons would probably not have become patients. Perseverance, chiefly in regard to occupation and perhaps therefore a compensation reaction, was noted in 68. Many of the same persons who lacked forcefulness and perseverance showed feelings of inferiority, which is compatible. However aggressiveness, which is often used to cover a feeling of inferiority, was more frequently noted in the ratio of 52 to 41 of the 105 persons, so undoubtedly some of that category were missed. Outward manifestations of sensitivity which were the criteria used were likewise not frequent, being checked in 40, though considering other characteristics and inadvertent expressions used in the discussion of other features this number probably is actually higher.

Mood levels considered when not under the influence of alcohol and taken from the statements of the patients and relatives gave 40 habitually cheerful, 27 depressed and 38 irritable. Almost all of the depressed and some of the irritable showed sufficient self anxiety to be classed as hypochondriacal. Some of the outwardly cheerful likewise evidenced hypochondriasis, which would here be classed as undue self interest, a form of narcissism. Manifest signs of the latter were much less frequent.

Psychosomatic features were not considered much in this study, as primary physical phases were purposely omitted. Anorexia was present in 48, a little less than half. Sleep disorder showed a high frequency, 81 evincing it. A pathologic response to alcohol in either physical or psychic sense was naturally present in all. Deterioration was thought to be present in 4 and enough indication was found to be questioned in 29. It would probably be observed more in the finer emotional range in this group.

One trait that has long been recognized as an integral part of alcoholic persons is superficiality. That applies as much for this group of professional and business people, socially adequate, with in general higher than average intellectual capacity, as it does for a group in a different social, economic or intellectual level. Ninety of the 105 were classed so. It is believed that in the present stage of knowledge about overindulgence in alcohol the most important question is "Why?" Why are these people superficial? Why, when they "get a thirst" do family, business, social responsibilities sit lightly? Why do entreaties of relatives, partners, friends do no good? The answer to these questions is well known to physicians. It embraces desire to conform, ambition to be able to drink socially, to be a man among men. All these are adolescent types of reaction indicative of emotional immaturity. They fit in with characteristics presented. So far as capacity, general activity and affect are concerned, most of these patients appear to fall into a cyclothymic category with associated mental mechanisms. Overdependence, feelings of inferiority and superficiality are more consistent with the schizoid tendency, with the last of these traits being predominant. The material offered here agrees with the opinion of many that the reason for alcoholism is to be found in the personality of the individual. As Kraines [6] says, "The addiction to alcohol is determined by the internal drive and not by the simple taking of alcohol." While one hesitates to make a didactic statement, the material seems to emphasize a lack of maturity and a difficulty in expression and accomplishment along some lines, chiefly in the emotional field of consciousness.

Parental attitudes have much to do with personality responses in the offspring. It would be far fetched as well as unfair to blame parents for development of alcoholic tendencies in children after the latter have been away perhaps for years. Nevertheless, in alcoholism and in other psychiatric disorders, notably schizophrenia, Williams' [7] description of "innocent yet dangerous parents" comes to mind with the carry-over into adult life of adolescent reactions affecting both parent and child, the latter even when grown up in other ways.

Therapeutic attempts in this group have been direct and educational. Since the disturbance is emotional, I believe with Miles [8] that the emotional component in treatment should be interjected as little as possible. The fact that these patients have in the main accepted business and social responsibilities is discussed with them. Various incapacities, maladjustments and conflicts are explained. Attempts at development of personal responsibility are then taken up. There is nothing new in this approach. It is one of the recognized forms of psychotherapy, considered outmoded by some, but in the light of the general intelligence level and general responsibility of this group it appears useful.

It is not possible to make an analysis of results in the same way that has been done for the material, for follow-up reports have not been made in many instances. Records indicate many notable failures, some worth while successes. It is felt that the characteristics and mental mechanisms described here in the study of individual patients have much to do with the personality reactions of the failures. A corollary to this may be that recognition on the part of the intelligent patient and a constructive attitude of personal responsibility developed through direct psychotherapeutic approach has had something to do with the successes.

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7. Williams, F. E.: Adolescence, Studies in Mental Hygiene, New York, Farrar & Rinehart, 1930, p. 32.

8. Miles, W. R.: Psychological Factors in Alcoholism, Ment. Hyg. 21: 529-548 (Oct.) 1937.

ABSTRACT OF DISCUSSION

DR. LLOYD H. ZIEGLER, Wauwatosa, Wis.: As physicians, we are likely to focus our attention on a few medical aspects of a person which are the basis for calling him a patient. Dr. Norbury has made a broader survey of the qualities of human nature in a group of reasonably successful and capable so-called nonpsychotic alcoholic patients. It would have been valuable to have available an equal number of nonalcoholic persons from similar strata of life for comparison. Had he studied a control group, some of the qualities of the alcoholic addicts that now seem significant would doubtless overlap with the controls to rob them of their seeming relationship to alcoholism. His patients were responsible about most things except themselves and their relationship to alcohol. One can see this in the diabetic patient who refuses to follow a diet restriction, in persons who hear the morning alarm clock but who refuse to heed it and so get to appointments late, in persons who violate the fishing or hunting laws or in automobile drivers who blame everything except themselves for accidents. This attribute is not limited to alcoholic addicts by any means. The fact remains that alcohol never of itself runs into any one's mouth. A definite person assumes responsibility for seeking it out; he uses his own right hand in lifting it to his mouth; he swallows it. These acts he performs and for them he must assume responsibility. What happens after this is often mixed up with excuses but not with responsibility. There is a dif-

ference. The alcoholic addict can ever point to the milieu of society which makes light of the affair. Judges of the court too who should be expert in placing responsibility have been known to use drinking as an excuse for leniency toward the results of the misuse of alcohol. Alcoholism is merely a name applied to the behavior of a definite person who thinks he can drink or let it alone but always drinks. This is a serious breach of responsibility to himself and often to his family. He becomes ex-alcoholic when he controls his irresponsibilities. Dr. Norbury's observation of superficiality interested me. Alcoholic addicts are often active, on the go, and may be bored by serious and sustained effort. They have seemed not quite grown up, living the part of youthful actors in a play world in which they get on splendidly together. There is great demand for the person who is witty and friendly, has warmth of personality, but who is not a deep thinker. Dr. Norbury's interest in these patients has been so broad that he has the armamentarium for succeeding where others might fail.

DR. G. WILSE ROBINSON JR., Kansas City, Mo.: Before the American Psychiatric Association in Richmond last month, Dr. Seligman presented a similar analysis. In Dr. Norbury's series such things as economic adjustment, success in occupation, intellectual level plus and social attributes plus are found in the majority of his patients. These are what we call normal characteristics. I have made this observation in my own cases time after time. I believe the problem as to why most economically successful alcoholic addicts drink is a rather simple one to state but a hard one to solve. He drinks for the same reason that the president of the bank, the circuit judge and the roustabout drinks: in order to have fun with other people or to relax. I believe that all alcoholic addicts start out as social drinkers, and it is only after alcoholism has become a problem that they attempt to hide it. Temptation to drink is almost universal. In certain social groups alcohol is served at every social function, and I have had many alcoholic patients tell me they get along fine until they attend some social event where liquor is served. I have had fellows tell me they have gone through the event and then the next day started to drink. From time to time it really becomes overwhelming, or their nervous fatigue becomes so intense that they need a sedative to produce relaxation. After they have started a drinking experience they cannot stop, and then they may drink in a solitary manner, hoping to hide it. I feel that the major problem with regard to the alcoholic addict is that he is no longer able to control alcohol. For many years he has been drinking socially. Suddenly he finds he cannot. The known diabetic patient is not tempted with sugar. Everybody understands why he cannot use sugar. However, the known alcoholic addict is not only served liquor but he is usually urged to drink for old times' sake. The strain on resistance is too much on many occasions. The problem of alcoholism will not be solved until we have found some way to make it possible for the alcoholic addict to drink again as he used to drink. I believe the problem is more physiologic than psychologic. My series includes a cross section of the social and economic life of Kansas City. It includes successes and failures, professional men and laborers, rich and poor, social registrants and bums. The only universal motif that I have been able to find is an inability to drink like other men. Certain thinkers may consider that psychologic. I consider it a physiologic problem, and I believe that the pathologic, physiologic and pharmacologic problem must be solved as well as the possible psychologic problems before we can find a solution to the problem of alcoholism.

DR. R. V. SELIGER, Baltimore: I congratulate Dr. Norbury on his fine paper on the personality characteristics of the alcoholic addict. I found that 50 per cent of the alcoholic addicts were extroverts and 50 per cent were introverts. As far as family inheritance is concerned, I think that one out of three of our group had a family background of alcoholism. I feel that the social drinker can handle alcohol so that it does not interfere with his important life activities, while a chronic alcoholic addict is handled by alcohol so that it does interfere with his important life activities. I do not feel that a chronic alcoholic addict can ever again drink. The only way for help and guidance and cure is total abstinence for life. Once a pathologic drinker, a person can never be a social drinker. There are about seven important factors in the successful rehabilitation of the alcoholic addict. Among these are the personality of the psychiatrist and the careful selection of patients. The patient has to have average intelligence or better. He has to have some emotional maturity in his make-up. His nervous system has to be undamaged at the time of treatment, and he must want treatment. You cannot treat a patient against his will. There has to be some psychiatric approach to find out what the mechanism is behind his drinking, if any, whether you go through the psychoanalytic approach or take the psychobiologic approach or any other approach. Then there must be a long period of rehabilitation, training and regulation of the patient's emotional life. Along with these there is a certain suggestive factor in which the patient knows that the doctor is interested inherently in alcoholic addicts, that he has helped others, and the patient sees by contact with former patients that alcoholic addicts can be helped. The follow-up for the rest of his life is important, either by correspondence or by visits. Finally it is my feeling that whether one considers that alcoholism is evidence of an escape mechanism or a neurosis or a psychosis or is evidence of latent or overt homosexuality or of a tendency to self destruction, one must realize that the situation is an individual psychobiologic personality reaction to life and life situations plus habit formation both in the emotional field and to the drug.

DR. F. GARM NORBURY, Jacksonville, Ill.: There are many ramifications of this subject. It was my purpose just to bring out certain features that had interested me in a personality study. I am inclined to agree with Dr. Seliger that the alcoholic addict cannot drink again. There are both psychologic and I think biochemical factors in that. If one can think of sensitization, the person who has used alcohol in the past is not only psychologically but I believe in a certain sense biochemically sensitized, at least in that it produces a catalytic type of explosive reaction; but that is rather outside the discussion of this particular paper.

STUDIES IN NICOTINIC ACID METABOLISM

V. A TEST FOR NICOTINIC ACID DEFICIENCY IN MAN

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It has not been possible to demonstrate nicotinic acid deficiency in man by the determination of either the blood level of nicotinic acid [1] or the level of urinary excretion.[2] The test described here is based on the level of excretion of nicotinic acid and trigonelline after a dose of nicotinamide.

In dogs maintained on a diet low in nicotinic acid it has been shown that a dose of nicotinic acid is largely retained.[3] After the dogs were well saturated by a high intake of nicotinic acid, most of the test dose of nicotinic acid was found in the urine as acid hydrolyzable nicotinic acid derivatives and trigonelline. This principle was applied to man in the following way:

Twenty-four normal adults were instructed to omit for three days coffee, all leguminous vegetables and

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A preliminary report was presented on July 24, 1941 before the Special Research Conference on Vitamins of the A. A. A. S. at Gibson Island, Maryland.

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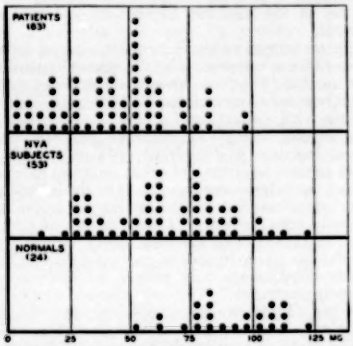
nuts (all of which contain trigonelline) from their otherwise adequate diets. On the evening of the second day the subjects voided just before dinner and discarded the urine. All urine, including that voided on rising, was collected until the next morning and preserved with



Distribution of extra excretion of total nicotinic acid after 500 mg. of nicotinamide (mg. in twelve hours).

toluene. Each subject ingested with the dinner 500 mg. of nicotinamide and again collected all urines until the next morning. The nicotinamide was administered intravenously to some of the patients as described later. No untoward or disagreeable reactions developed in any of the subjects after the oral or intravenous administration of 500 mg. of nicotinamide.[4] The control and test urines were analyzed for the acid hydrolyzable nicotinic acid derivatives and for trigonelline by the method previously published.[5]

In addition to the 24 normal subjects the test was applied also to two other groups, the first consisting of 53 young men (aged 16 to 24) as they were admitted to a local National Youth Administration camp for the purpose of physical upbuilding by means of diet and exercise. This group consisted of underweight and undernourished boys, physically under par, with or without correctable defects but capable of physical work. There were no cases of organic or infectious disorders.[6] The boys all came from the lower economic levels of the local population, as did the subjects of the next group of hospital patients.

Sixty-three patients, unselected as to nutritional status, type or duration of disease or age, were also subjected to this test. Of this group 50 represented every patient, without exception, who was admitted to a male public medical ward of Duke Hospital over a five week period. The other 13 (6 men and 7 women) were recently admitted medical patients taken at random. The tests were started on all of these patients within two days of their admission to the hospital while they were on the regular ward diet. They were all acutely or chronically ill with the large variety of diseases found in a public medical ward. Apart from 1 case of partly treated pellagra and 4 cases of frankly characterized nutritional deficiency, the incidence of symptoms of general malnutrition such as loss of weight, anorexia and secondary anemia was relatively high in this group, being partly due to its low economic level and partly inherent in the clinical conditions which they presented.

A comparison of the findings in these two groups with the group of normal subjects is shown in the accompanying table and chart.

It will be observed that no significant differences in the nicotinic acid excretion were found in the control urines of the three groups. However, marked differences in levels of excretion of these groups appeared in the urines following the ingestion of 500 mg. of nicotinamide. These differences were found almost entirely in the trigonelline fraction of the urine. This explains the results of Briggs,[7] who determined only acid hydrolyzable nicotinic acid derivatives and not trigonelline and was therefore unable to find differences between normal and deficient subjects. The extra total nicotinic acid (including trigonelline) excreted after the test dose averaged 90 mg. for normal persons, 63 mg. for NYA subjects and 41 mg. for hospital patients.

The distribution of individual value for the extra total nicotinic acid after the test dose is shown in the chart. All of the normal subjects gave values above 50 mg. and 84 per cent were above 75 mg., while among the patients only 11 per cent were above 75 mg. and 54 per cent were below 50 mg. The NYA group occupied an intermediate position between these two extremes.

The question of the completeness of absorption of the nicotinamide arose, and it was found that in 4 normal subjects the results obtained after intravenous and oral administration of the test doses, given several days apart, agreed very well. In 12 of the patients in whom there were symptoms of gastrointestinal or liver disease the amide was administered intravenously and the test was repeated orally one to four days later. With the exception of 1 subject in whom the oral test was considerably lower than the intravenous, the results were similar to those obtained with successive oral doses. However, in diseases which may affect absorption from the gastrointestinal tract, including severe deficiencies, it is advisable to give the test dose intravenously. The results of the intravenous test only were included for these 12 patients in the table and chart.

Twelve Hour Urinary Excretion of Nicotinic Acid and Trigonelline in Three Groups of Subjects Before and After 500 Mg. Dose of Nicotinamide

	Nicotinic Acid *		Trigonelline		Total †	
	Range, Mg.	Average, Mg.	Range, Mg.	Average, Mg.	Average, Mg.	Extra Excretion After Test, Mg.
Normal persons (24)						
Before	0.5- 1.5	0.9	5 - 21	10	9.9	
After	3 -19	7.3	76 -136	104	100	90
NYA subjects (53)						
Before	0.4- 1.8	0.8	3.8- 17.6	8.3	8.3	
After	0.5- 4.4	1.9	19 -141	77	71	63
Hospital patients (63)						
Before	0.1- 2.3	0.7	1.3- 28	7.7	7.7	
After	0.3-23	2.7	2.6-109	51	49	41

* Includes all nicotinic acid derivatives hydrolyzable by boiling with 6 normal hydrochloric acid.

† Total represents the sum of nicotinic acid and trigonelline calculated in terms of nicotinic acid.

4. The nicotinamide used in this study was the gift of the Upjohn Company.

5. Perlzweig, W. A.; Levy, E. D., and Sarett, H. P.: Nicotinic Acid Derivatives in Human Urine and Their Determination, J. Biol. Chem. **136**: 729 (Dec.) 1940. Sarett, Huff and Perlzweig.[3]

6. Dr. I. H. Manning Jr., medical director, and the staff of the NYA camp cooperated in this work.

7. Briggs, A. P.: Excretion of Nicotinic Acid in Pellagra, Proc. Soc. Exper. Biol. & Med. **46**: 374 (March) 1941.

SUMMARY

The majority of hospital patients admitted to a general medical ward excreted in twelve to fourteen hours much less of a large dose of nicotinamide than normal persons. A group of undernourished youths excreted an intermediate amount after the same dose. The procedure described may serve as a test for nicotinic acid deficiency in man.

UNCOMPLICATED FRACTURES OF RIBS AND MAJOR INJURIES OF THE CHEST WALL

TREATMENT BY INFILTRATION WITH LOCAL ANESTHETIC

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AND
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The usual attitude of the physician about fractures of the ribs is well summarized by the following quotation, taken from a current textbook on the treatment of fractures:[1] "Fractures of the ribs in themselves are not of much moment, and it is only their complications that make them important." The frequency of fractures of the ribs, together with the relative effectiveness of treatment afforded by wide hemicircumferential strapping of the chest, has resulted in widespread acceptance of this method of treatment. Whatever discomfort arises has been passed over as being "part of the treatment." However, therapeutic procedures should be made as agreeable to the patient as possible and the course of convalescence, even from minor injuries, free from pain and complications. In a recent authoritative symposium on fractures[2] the only method of treating fractures of the ribs given was strapping of the chest with adhesive tape.

Our experiences with the treatment of sprains, marginal fractures and painful myofascial disorders with procaine hydrochloride have led us to apply this method to other painful conditions. Investigators[3] who have had the opportunity to witness the shorter and milder course of patients suffering from the acute traumatic conditions mentioned have advocated this method as the treatment of choice. At first we used it hesitantly on patients who suffered only pain from local and isolated fractures of ribs, but later we extended it, with the same immediate relief from pain and general alleviation of the course of convalescence, to patients with the major varieties of injuries to the chest that are described in detail later in this paper. While the results of treatment with procaine hydrochloride alone were satisfactory, the adoption of a double solution containing 0.5 per cent procaine hydrochloride and 0.1 per cent eucupine dihydrochloride (isoamylhydrocupreine dihydrochloride), to which epinephrine hydrochloride (10 drops per hundred cubic centimeters) was added, gave more prolonged primary anesthesia (three or four hours, as compared to one-half to one hour). This double solution is now used routinely in treating thoracic injuries at the Guthrie Clinic and Robert Packer Hospital.

The advantages of the injection treatment for fractures of ribs as compared with the immobilization treatment are that (1) pain is immediately abolished, because of the removal of pleural and local afferent sensations; (2) the vital capacity and the tidal air remain at a more normal level; (3) excessive coughing due to pleural irritation is diminished; (4) as a result, the use of general sedatives and cough mixtures is lessened or made entirely unnecessary, and (5) the injection is a minor procedure that can be carried out in the office. The only drawbacks are (1) the danger of infection, which is minimal with adequate aseptic technic (infection did not occur in any of our cases) and (2) the psychic disadvantage with persons who "do not like injection treatments." The only absolute contraindications are (1) known hypersensitivity to procaine or epinephrine hydrochloride and/or sensitivity to the quinine series of drugs (minor untoward reactions can be abolished by giving sodium amytal in doses of 1½ to 3 grains [0.09 to 0.18 Gm.] one hour before injection) and (2) a dirty or infected open wound in the skin that cannot be blocked off from the field of injection.

On reviewing the literature, we found that Zoppi[4] has been the only author to recommend local infiltration of procaine hydrochloride for fractures of the ribs. In his article the daily infiltration of the analgesic was recommended. A number of articles have given supporting evidence to the effectiveness of regional nerve block in both simple and complicated injuries to the chest. Alcoholization of the intercostal nerves has been advocated by Latteri,[5] Rabboni[6] and Grieco,[7] the method having been originated for the control of pain from tuberculous pleuritis. In this country only Rovenstine and Byrd[8] have utilized intercostal regional anesthesia for analgesia in cases of extensive thoracic injuries. These authors expressed the belief that the method lessens the mortality, contributes greatly to the patient's comfort and is important as a prophylactic agent against respiratory infection.

MANAGEMENT

After the diagnosis is made and the site of the fracture accurately localized by roentgenograms, the patient is placed on a table with the site of fracture in a prominent and accessible position. A mark is placed on the skin with a solution of gentian violet, a silver nitrate stick or some other coloring agent that can be readily distinguished from the cutaneous disinfectant. The surrounding area is prepared with half strength tincture of iodine and draped with sterile towels. For convenience, in our clinic a standardized tray setup, containing syringes, needles and the solution to be injected, is prepared on the operating floor and transported to the

From the Department of Orthopaedic and Traumatic Surgery, the Guthrie Clinic and the Robert Packer Hospital.

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5. Latteri, S.: Alcoholization of Intercostal Nerves in Therapy, Riv. san. siciliana **21**: 249, 1933.

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outpatient clinic or to the bedside. The solutions of procaine hydrochloride and eucupine dihydrochloride are sterilized separately in 1 per cent strength and mixed by diluting with physiologic solution of sodium chloride to obtain a final concentration of 0.5 per cent of the former and 0.1 per cent of the latter. Epinephrine hydrochloride solution (1:1,000) is then dropped into this mixture. A wheal is raised over the proposed site of injection, 1 cc. of solution is placed deeply in the subcutaneous tissues and 2 or 3 cc. of solution is infiltrated directly into the site of fracture. If this site and its surrounding hematoma cannot be directly entered or identified with the tip of the needle, 4 to 6 cc. of solution is placed in the surrounding tissues. With fractures above and including the third rib and with posterior fractures, especially those covered by the scapula and its mesial muscular attachments, it is difficult to make an injection directly into the site of fracture. Elsewhere localized tenderness and crepitus will serve as a guide to accurate deep placement of the needle. Single fractures in many ribs, multiple fractures in the same rib, extensive injuries to the thoracic wall, mild to moderate shock and the presence of emphysema instead of being contraindications are, indeed, indications for use of the method. A small gauze dressing or a small cotton or gauze pledget soaked with collodion serves as a temporary dressing. Often one injection suffices, but in 8 (25 per cent) of the 32 cases of minor fractures covered in this report it was necessary to perform another injection at the site of fracture in one to four days. In no instance was a third injection necessary. Relief from pain is difficult to estimate and is impossible to measure exactly, but the normal response from the patient was that he was "comfortable," being immediately relieved from pain and only "slightly uncomfortable" for two to five additional days, with only an occasional pain on sudden positional change for two more weeks. The average period of disability (until the patient returned to his former occupation, usually light manual labor) was four and two-tenths days for the 32 patients. Twelve with fracture of a single rib returned to work immediately after treatment and were not "disabled." The 32 patients were given entirely ambulatory treatment.

In addition to mentioning the cases of minor fractures of ribs we wish to illustrate the use of infiltration with local anesthetic for major injuries to the chest wall by the following case reports.

REPORT OF CASES

CASE 1.—*Open operative excision of protruding ends of ribs that had punctured the pleura; subsequent use of procaine hydrochloride-eucupine dihydrochloride solution to relieve pain in the chest from multiple fractures of ribs.*

C. F., a man aged 39, received a severe injury to the chest by being stepped on by a horse. When examined shortly after the injury had taken place he had pain along the whole right side of the chest. He was dyspneic and was in mild shock. Examination revealed dulness of the chest up to the seventh rib and exquisite tenderness in the right posterior scapular line. Roentgenographic examination revealed fractures of the right second to eleventh ribs inclusive, which were on the average 9 cm. from the costovertebral junction (figs. 1, 2 and 3).

On admission, the blood pressure was 116 systolic and 70 diastolic. The blood contained 3,400,000 red cells and 16,000 white cells per cubic millimeter. Since roentgenograms of the chest showed central displacement of the right fifth rib into the chest wall and through the pleura, it was deemed necessary to examine this rib at open operation.

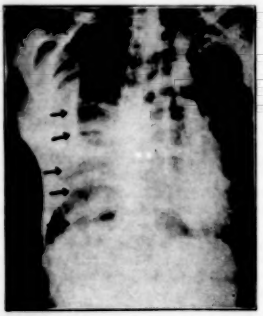

Fig. 1.—The chest in case 1 on the day of injury. Open operation was performed to excise sharp ends of ribs that had lacerated the costal wall, including the pleura. The arrows indicate the free fragments at the sites of fracture of the fourth and fifth ribs.

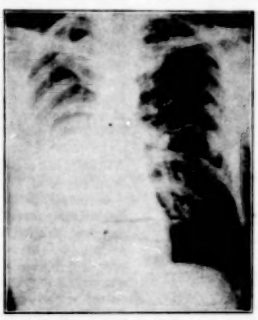

Fig. 2.—The chest in case 1 seventeen days after operation. Note the fluid level and the shift of the costal wall after excision of fragments of the fifth and sixth ribs.

Fig. 3.—The chest in case 1 two months after injury. The right side had practically cleared.

Accordingly this was done, with infiltration anesthesia with procaine hydrochloride. The costal wall from the third to the seventh ribs was bared through a posterior horizontal incision which allowed the scapula to be pulled laterally. The mesial end of the right fifth rib was found to have perforated the pleura. One-half inch was resected from both the mesial and the lateral ends of the fractured fifth and sixth ribs, the pleural wound closed en bloc with the adjacent intercostal muscles, the rhomboid muscles reapproximated and the wound closed. Physiologic solution of sodium chloride was administered intravenously throughout the operation, which was well tolerated by the patient. The blood pressure at the termination of the operation was 100 systolic and 74 diastolic. Into each of the remaining fractures, not visualized at operation, 3 cc. of a 1 per cent solution of procaine hydrochloride was injected. The patient returned to the ward less dyspneic and breathing without pain in the chest. The vital capacity (normal, 4.4 liters) prior to operation was only 1,200 cc. Immediately after operation it rose to 1,600 cc.

On the morning of the following day the patient was fairly comfortable, but pain in the chest increased and the respiratory rate increased from 28 to 40. He was placed in an oxygen

tent sixteen hours after operation. Throughout this day the blood pressure was well maintained and the red blood cell count remained at 3,900,000 per cubic millimeter. In spite of the use of the oxygen tent, the patient became more dyspneic on the third postoperative day, and the slight cough which he had had on the second day increased in annoyance and severity. The cough was not productive, and the patient suffered extreme pain with each attempt at coughing. He was gravely ill, and the temperature, which had previously been normal, rose to 101 F. Multiple injections of 3 to 4 cc. of a solution of 0.5 per cent procaine hydrochloride and 0.1 per cent eucupine dihydrochloride were made as nearly as possible into the site of each fracture (forty hours after the operation). The patient was relieved from pain immediately, and within fifteen minutes his cough became productive of plain and faintly blood-streaked sputum. The respiratory rate declined from 40 to 32.

Preoperative and Postoperative Estimation of Vital Capacity After Injection Treatment for Fractures of Ribs and Injuries to the Chest

Case	Normal, Liters	Prior to Injection, Liters	Immediately After Injection, Liters	Final Determinations: Interval	Final Determinations: Amount, Liters
1	4.4	1.2	1.6	8 mo.	3.9
2	4.5	0.4	1.5	2 mo.	3.3
3	4.4	1.5	2.0	4 mo.	3.6

From this point, the entire clinical picture shifted. The temperature declined to an average of 99 F. It was not again necessary to inject the solution into the fractured ribs, although moderate pain returned as the patient was able to expectorate collections of mucus from the upper respiratory tract. During the ensuing twenty-three days' stay in the hospital, the vital capacity slowly rose to 2 liters. Further increase was hampered by the collection of fluid in the right side of the lower part of the chest up to the sixth rib. Since the patient was not dyspneic except on moderate exertion, there was no indication for aspiration of the fluid. He continued to improve after leaving the hospital, but the vital capacity did not return fully to normal for two months, and the fluid did not all disappear from the right side of the chest until that time.

When last seen, eight months after the injury, he was working daily at moderately heavy manual labor without pain or dyspnea. Roentgenograms showed the chest clear, and the vital capacity was 3.9 liters (table).

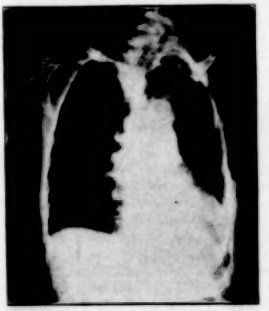

Fig. 4.—The chest in case 3. The arrows indicate the sites of fractures of ribs. Note the mottling over the left side of the upper part of the chest produced by subcutaneous emphysema.

CASE 2.—*Complete and permanent relief from pain in simple multiple fractures of ribs.*

H. A. B., a woman aged 55, sustained contusions to the left shoulder and thoracic region from direct trauma in an automobile accident. When she was first seen rib strapping had been applied as an emergency measure elsewhere but had only partially relieved pain. Roentgenographic examination showed fractures of the eighth, ninth, tenth and eleventh ribs in the left anterior axillary line. When admitted, the patient was suffering severe pain in the left side of the chest and was unable to take a deep breath because of it. Severe pain was also present on movement of the left shoulder, but roentgenographic examination revealed no bony injuries there. Anesthesia of the painful areas in the ribs was obtained by depositing 3 cc. of solution directly into each site of fracture. Relief was obtained immediately and persisted during the patient's remaining four days in the hospital. On discharge, the patient stated that the only pain she had had while in the hospital had been associated with movements of the shoulder or had been produced by sudden rising or twisting. Ordinary breathing efforts and motion had been entirely without pain.

CASE 3.—*Fractures of ribs complicated by subcutaneous emphysema.*

M. S., a frail woman aged 82, was involved in an automobile accident, suffering injuries to the left shoulder and the right side of the chest. She was admitted to the hospital on the morning after the accident. A local physician had strapped her chest, but this had not relieved the pain. She had not eaten the previous day because any movement of the neck, including deglutition, caused severe pain in the chest. She was likewise unable to turn herself in bed for this reason.

Fig. 5.—The chest in case 4. The arrows indicate the fractures of ribs in the left midaxillary line.

Roentgen examination revealed fracture of the second and third ribs near the left sternocostal margin and fracture of the right pubic ramus (fig. 4). There was considerable subcutaneous emphysema over the left side of the chest, which spread out over the left side of the neck and the left axilla. The patient was given a single injection of 10 cc. of a solution of procaine hydrochloride and eucupine dihydrochloride, this quantity being equally divided between the sites of fracture on the second and third ribs. From this time, the patient was able to sit in a semireclining position in comfort and to turn slightly in bed in accordance with preventive measures taken to prevent passive congestion, and she could take fluids and food orally without discomfort. The relief was instantaneous after injection and continued until the following day, when only slight discomfort in the chest returned. During the remainder of her hospital stay she was comfortable. The air beneath the skin was gradually absorbed, so that none was present ten days after injury.

CASE 4.—*Fracture of four ribs in the left midaxillary line, with incomplete fracture of two of the ventral ends of the same ribs; hemopneumothorax and subcutaneous emphysema.*

G. M., a man aged 58, was struck by an automobile while walking on a highway and was brought immediately to the hospital. He had pain in the chest and expectorated a fair amount of bloody sputum. There was extensive subcutaneous emphysema over the left thoracic wall, the left side of the chest was hyperresonant and there were no breath sounds. Roentgenograms showed fractures of the fifth, sixth, seventh and eighth ribs in the left midaxillary line (fig. 5), incomplete fracture of the vertebral ends of the sixth and seventh ribs and an undisplaced fracture through the left pubis. A complete pneumothorax was visualized on the left. The vital capacity was but 0.4 liter (normal, 4.5 liters), but the test was unsatisfactory because of the extreme pain. He received an injection of 10 cc. of procaine hydrochloride-eucupine dihydrochloride solution into the site of fracture in the left midaxillary line and an additional 5 cc. into two contused areas in the right side of the chest, with immediate and permanent relief from pain. The patient volunteered that during the next week he was uncomfortable only on quick turning in bed. He was kept

in bed for ten days because of the complete pneumothorax. Periodic roentgen examination of the chest demonstrated fluid up to the tenth rib posteriorly and partial expansion of the left lung in ten days. When the patient was last seen, ten weeks after injury, the vital capacity, which for the first ten days after injury had remained between 1.4 and 2.3 liters, had risen to 3.3 liters. All symptoms had subsided.

CASE 5.—*Sternomanubrial dislocation.*

M. C., an obese man aged 46, injured the chest by direct impact of the body of the sternum against the steering wheel of an automobile. This resulted in tearing of the sternal manubrial ligaments, with anterior dislocation of the manubrium on the body of the sternum. Fracture of the right fourth, fifth, sixth and seventh ribs in the anterior axillary line also occurred (figs. 6 and 7). He was admitted to the hospital on the morning following the accident.

Physical examination at that time failed to reveal evidence of any trauma except that of the thoracic area. The patient was suffering considerable pain, and respirations were shallow and difficult because of it. He was immediately given an injection of 15 cc. of a solution of procaine hydrochloride and eucupine dihydrochloride into the site of dislocation. Five cc. of the solution was deposited with a long needle about the site of each fracture. The respiratory rate slowed from 40 to 28, the breathing became correspondingly deeper and the patient was immediately relieved from pain. The relief continued from noon until 10 p. m., at which time the patient complained of a return of discomfort, mainly at the site of dislocation in the anterior midline of the chest. He was given another injection of 20 cc. of a solution of procaine hydrochloride and eucupine dihydrochloride and passed a fairly comfortable night. From this point, thirty-six hours after the accident, the patient was fairly comfortable and required no injections.

During the remainder of his hospital stay, ten days, he suffered slight discomfort at the site of dislocation but in the main was free from the exquisite pain from which he had suffered on entrance. He was advised to spend several weeks in recumbency and extension because of the tendency for the superior fragment to be displaced. This advice was unheeded, and he insisted on leaving the hospital. Pain had not been a prominent feature of his course from the second day in the hospital. He began to experience increasing pain in the back at the fourth and fifth thoracic segments and returned to the hospital for open reduction and fixation of the dislocation two months after the original injury.

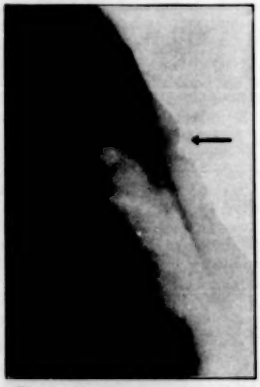

Fig. 6.—Lateral roentgenogram of the chest in case 5. The arrow points to the sternomanubrial dislocation. This view was taken forty-eight hours after injury. The lower fragment is depressed.

COMMENT

The cases reported in this paper demonstrate the immediate and effective relief from pain obtained after minor fractures of the ribs (in 32 cases) and after major injuries to the chest (in 5 instances) through infiltration of a solution giving prolonged local anesthesia. When the precautions usually taken before the injection of a local anesthetic are observed, there is as much reason for the injection of this solution directly into the site of fracture in ribs as there is for the use of a local anesthetic before the reduction of fractures of the long bones. This method eliminates rib strapping and sedatives, both of which exert unfavorable effects on thoracic injuries. Strapping the chest reduces the effective excursion of the chest wall and thus reduces tidal air and vital capacity and lessens the effectiveness of the cough reflex. Sedatives, especially drugs of the opiate series, depress respirations, decreasing pulmonary ventilation, and diminish the sensitivity of the cough reflex. Smaller doses of these drugs, which will not have the untoward effect mentioned, usually are not effective in controlling pain.

Fig. 7.—The end result in case 5, three months later. Note that reduction has been maintained.

A more logical means of controlling pain is to interrupt the painful afferent stimuli at their origin at the site of fracture. To do this is compatible with the known effectiveness of local anesthetic agents in the treatment of sprains and other areas subjected to trauma. While fractures of the ribs and major injuries of the chest wall can be treated either by local infiltration of the anesthetic agent or by the production of regional anesthesia by paravertebral block, the latter is technically more difficult, and the end results are the same.

The comfort of all the patients on whom the method was used improved after the injection. Indeed, the method was considered life saving in 1 instance (case 1), since the entire clinical picture shifted after the use of the solution.

All authorities are agreed that injuries to the thoracic wall, including penetrating wounds, are best managed conservatively. Boland [9] and Elkin [10] reported a mortality of only 8.5 per cent from all types of pulmonary and pleural injuries in 1,009 cases in which such injuries presented most, if not all, of the clinical picture. The indications for operation for such injuries are usually extensive laceration of the pleural wall, with or without hemorrhage, and hemorrhage from the tearing of an intercostal artery. The occurrence of tension pneumothorax calls for careful aspiration of blood and air from the pleural space. Aspiration as a rule should be delayed twenty-four or forty-eight hours if possible, since a pleural tamponade will succeed in controlling hemorrhage in a certain number of cases. A review of methods and indications for treatment may be found in the article by Berry [11] and in the textbook of Graham, Singer and Ballou.[12]

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10. Elkin, D. C.: Wounds of the Thoracic Viscera, J. A. M. A. **107**: 181 (July 18) 1936.

11. Berry, F. B.: Wounds of the Thoracic Viscera, Am. J. Surg. **39**: 12, 1938.

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SUMMARY AND CONCLUSIONS

The use of a local anesthetic with a prolonged action in 32 cases of minor fractures of ribs and 5 major thoracic injuries eliminated the necessity of strapping the chest wall with adhesive tape and the excessive use of sedatives to control pain. A more physiologic state was thus preserved throughout convalescence. It is our clinical impression that the comfort of the patient is greater with this type of treatment than with any other.

LEPTOSPIRAL INFECTION (WEIL'S DISEASE) AS AN OCCUPATIONAL HAZARD

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Human leptospiral infections have been frequently associated with circumstances or occupations in which the patient has had contact with the excreta of rats or, rarely, with those of dogs. Isolated cases have been adjudged industrial accidents, and compensation has been awarded in this country and abroad. Recent investigations have shown an unsuspected incidence of the infection in certain occupations. This finding has increased the concern about the industrial liability that may be involved.

Leptospiral infections have been confused clinically with catarrhal jaundice and other diseases that have no occupational association. It becomes essential, therefore, to establish the diagnosis by laboratory methods. In this country at the present time a leptospiral infection is but rarely considered in the differential diagnosis until the disease has reached a critical phase, and even then laboratory facilities are so unavailable and methods so unfamiliar that a conclusive diagnosis is usually remote. It may be concluded that a leptospiral infection should be considered in the differential diagnosis of any illness of a patient whose occupation or circumstances bring him into contact with rat or dog excreta and that laboratory facilities and methods should be refined to insure a correct diagnosis. It is also important that workers and their employers should be informed of the potential hazard of material contaminated by rat and dog excreta and that precautions should be taken to prevent such contamination.

EPIDEMIOLOGY

Incidence.—Leptospiral infections have been recognized more extesively and with increasing frequency[1] since the etiologic agent was first demonstrated by Inada and his colleagues in 1914. For example, in the Netherlands between 1924 and 1938 there were 852 reported cases.[2] Among those of known source, 257 cases (78 per cent) followed swimming or water accidents and 68 (22 per cent) were of occupational origin. Of the 68 patients 16 were fishermen, half of the 52 others were engaged in work that had to be performed on or in water (divers, seamen, reed cutters and so on) and the other half were occupied in places overrun with rats (sewers, slaughter houses, butter factories, barns and stables). Thus, in 91 per cent of the cases infection followed contact with water contaminated with rat urine, and in the other 9 per cent there was contact with moist materials similarly contaminated.

In Great Britain between 1933 and 1939 there were 246 authentic cases of leptospiral infection.[3] Of these, 144 (58.5 per cent) were in fish workers, 34 (13.8 per cent) in coal miners, 21 (8.5 per cent) in sewer workers, 16 (6.5 per cent) in swimmers, and the remainder were associated with at least seventeen occupations or circumstances. During a single year beginning in October 1933, 138 cases among sugar cane workers in Australia were reported.[4] Of the 1,232 specimens of blood from persons suspected of suffering from leptospiral infection examined at the Pasteur Institute in Paris during 1933, positive agglutination tests were obtained in 23.1 per cent of the cases.[5]

Seventy-three cases of leptospiral infection in North America that seem authentic are tabulated in the present report. In addition, about half that number were reported as leptospiral infections by the respective authors, but in these the laboratory proof is not regarded as adequate. Several outbreaks of infectious jaundice in which leptospira organisms were implicated only by circumstantial evidence are omitted from our series.

TABLE 1.—*Yearly Distribution*

1905	1	1934	3
1921	1	1935	3
1924	2	1936	3
1925	2	1937	6
1926	5	1938	22
1927	1	1939	18
1928	1	1940	2
1932	1		

The first case of authentic leptospiral infection was that of Stimson.[6] He described an organism in silver-stained sections of the kidney of a patient who died in 1905 during a yellow fever epidemic in New Orleans. It was not until recently, however, that this organism, which Stimson called ? Spirochaeta interrogans, was recognized to be identical with Leptospira icterohemorrhagiae. Now it has been suggested that the term Leptospira interrogans deserves precedence.[7] The second reported case occurred in Albany, N. Y., in 1921. This was a laboratory infection that was recognized immediately.[8]

The authentic cases of leptospiral infection in North America are arranged in tables 1, 2 and 3 according to the year, season and locality in which they occurred.

Leptospiral infections are more severe in older patients and far more common in males than in females. These facts are illustrated in a comparison of the Amer-

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The present report is based on part of a thesis on leptospirosis by the senior author that received the Taylor Instrument Company award of the Rochester Academy of Medicine for 1941.

1. Alston, J. M., and Brown, H. C.: The Epidemiology of Weil's Disease, Proc. Roy. Soc. Med. **20**: 741 (Feb. 26) 1927; Lancet **1**: 569 (March 6) 1937.

2. Walch-Sorgdrager, B.: Leptospiroses, Bull. Health Organ. League of Nations **8**: 143, 1939.

3. Davidson, L. S. P., and Smith, J.: Weil's Disease in the North-east of Scotland, Brit. M. J. **2**: 753 (Oct. 14) 1939.

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5. Erber, B.: La spirochétose ictérohémorragique en France en 1923 à 1932, d'après la statistique des épreuves d'agglutinations effectuées à l'Institut Pasteur, Bull. Off. internat. d'hyg. pub. **26**: 1737 (Oct.) 1934; Brit. M. J. **2**: 1155 (Dec. 22) 1934.

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ican series with that of the Netherlands,[2] as shown in table 4. In the American series the mortality is 17 per cent in patients less than 40 years of age, 63 per cent in patients more than 40 years of age and 33 per cent in the entire group. In the Netherlands series the mortality is 7 per cent, 33 per cent and 12 per cent, respectively, for these groups. The higher mortality in the American series is explained in part by the fact that mild forms of leptospiral infection are often overlooked in this country.

The circumstances or occupations involved in the American series are shown in table 5.

Mode of Infection.—Leptospiral infection is considered to be possible through the intact skin and mucous membranes as well as through these tissues after injury. Usually it follows contact of the abraded or sodden skin with infected mud or water, but it may follow the inhalation of water or the bites of rats, dogs or ferrets.[1] One patient who was found to have a latent leptospiral infection said that he had been bitten by rats on several occasions during the eighteen years that he had lived on a city dump. Four other persons who were severely bitten by rats gave no evidence of a leptospiral infection. White rats may become carriers of leptospira organisms and have been responsible for human infections.[9] One instance has been mentioned in which infection resulted from stepping with an abraded foot on the blood of a rat that had just been killed.[10] Mechanical transmission of the infection has occurred, as illustrated by a case resulting from the bite of a dog that had previously killed a rat and by two other cases following the bites of ferrets that had had contact with rats.[11] In another instance the macerated tissue of a rat that had been caught in a dairy machine was spattered in the face of the patient. Occasionally, association of human cases with infected dogs has been reported.[12] Contact with a jaundiced pig has been mentioned.[13] One case attributed to coitus has been recorded[14] and another considered to be probable in the American series. It has been suspected that contact with postmortem tissues has been responsible for several cases.[15] A number of laboratory infections have been known;[16] among them two are included in the American series.

The vast majority of human leptospiral infections have originated from water or moist objects contaminated with infected urine. In most countries it has been shown that the kidneys of about 20 per cent of the wild rats are infected with leptospira organisms.[2] Infected dogs also have been recognized as chronic shedders of pathogenic leptospira organisms.[17] White rats,[9] gophers,[18] foxes,[19] ferrets,[20] guinea pigs[21] and other animals have been infected experimentally or found to be diseased naturally. They must therefore be considered as potential vectors. The urine of infected human beings has also been regarded as hazardous in the spread of the disease.[22]

Virulent leptospira organisms have been found to be viable for at least twenty-two days in surface waters,[23] and they have been maintained in artificial mediums for five hundred and eighty-five days.[24] Leptospira organisms of debatable pathogenicity for man have been found widely distributed in water, slime and mud.[25] Water-borne epidemics have been usually associated with submersion in the water, although one unproved drinking water epidemic has often been quoted.[26] Acid soils and water (p_H less than 6.8) have rarely been found to contain leptospira organisms;[22] likewise leptospira organisms have rarely been found in soil or water having a concentration of salt above 0.17 per cent.[1] That the organism quickly dies in an acid medium, in salt water and in strong sunlight is in accord with the known distribution of the human disease.[1]

TABLE 2.—*Seasonal Distribution*

January	1	July	12
February	4	August	10
March	4	September	9
April	1	October	9
May	2	November	9
June	7	December	2
Unknown	3		

TABLE 3.—*Geographic Distribution*

New York	20	Maryland	2
Ohio	11	New Jersey	2
California	9	Connecticut	1
Pennsylvania	9	Colorado	1
Michigan	7	Washington, D. C.	1
Canada	4	Louisiana	1
Virginia	2	Massachusetts	1
West Virginia	2		

Compensation Awards.—In Germany during 1922 or thereabouts a man developed a fatal leptospiral infection two weeks after falling into a cesspool. This case was analogous to one already published by Stirl. In both instances the right to workman's compensation was recognized.[27] Another case was reported in 1927, that of a man who fell into a ditch, swallowed some of the

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water and developed a fatal leptospiral infection.[28] A connection between the accident and the man's death was affirmed by the court. Some of the 23 cases occurring between 1926 and 1932 in Hamburg were regarded as accidental occupational infections. As examples, 2

TABLE 4.—*Mortality, Age and Sex Distribution*

	American Series	Netherland Series
Total number of cases	73	374
Age groups:		
Less than 10 years	0	11
10 to 40 years		
Males	44	187
Females	4	23
40 to 60 years		
Males	20	42
Females	0	7
More than 60 years		
Males	4	15
Females	0	1
Unknown		
Males	1	79
Females	0	9
Deaths		
Males	24	42
Females	0	2
Children	0	0
Mortality, per cent		
Under 40 years	17	7
Over 40 years	63	33
Total	33	12

men who worked in the drains, a dock laborer working on a dredger in the harbor and 2 other cases among the crew of a ship overrun with rats may be cited.[29] In the fatal case of the Hamburg dock laborer it was necessary to pay compensation.

A legal precedent was established in England in 1925, when compensation was awarded for the death of a coal worker who had a leptospiral infection.[30] The award was contested in the Court of Sessions but dismissed. The case in question was *Raeburn* v. *Lochgelly Iron and Coal Company, Ltd.* (20 B. W. C. C., 637).

In England the Departmental Committee on Compensation for Industrial Diseases in its third report, 1935, came to the following conclusions:

> After reviewing the evidence the Committee recommends that infection by S. icterohemorrhagiae should entitle a workman to compensation for disablement, but only if confirmed by bacteriological or serological examination. In fatal cases, pathological evidence of the infection should be accepted. In the event of the disease becoming more widespread or of other developments, it may be found that it ought no longer be scheduled as an industrial disease.[31]

The committee found that a recent and acute infection can be diagnosed either serologically by a high or ascending titer or bacteriologically by demonstrating the leptospira in the blood or urine, directly or by animal inoculation. It is difficult to prove that death is caused by the disease, because the changes found after death are not specific; moreover, a patient may die in the second week, before material for laboratory examination has been obtained. Nevertheless, the committee is satisfied that it is reasonably certain that death was caused by the disease if the claimant can show morbid changes compatible with it, an appropriate history and clinical picture, and the absence of any other recognizable cause of death. Under the Act, the certifying surgeon must examine the workman personally, but the committee considers that it should rest with the workman or some one acting for him to produce the laboratory evidence in support of his application.[32]

In England, in an arbitration heard by Judge Dumas at the Westminster County Court on May 17, 1935,[33] £600 compensation was awarded the widow and three young children of a sewer worker who died of a leptospiral infection. On June 29, 1936 an award of £435 6s. was paid the widow of another sewer worker who died of the infection. Testimony presented at the Clerkenwell County Court revealed that the plaintiff abraded his knuckles while using a chisel in the sewer during the early part of August 1935. For a few days nothing was thought of the injury but he became ill, stopped work during the latter part of the month and died on September 11. Leptospira organisms were isolated from the patient's blood by the inoculation of a guinea pig. It was brought out by the attorney for the plaintiff that both the injury and the infection occurred while the patient was working but that compensation was entitled whether the injury occurred at work and the infection elsewhere or the injury elsewhere and the infection at work. As to notice of injury being reported to the company, which was a crucial question in the case, it was ruled that it was given "as soon as practicable after the accident," even though it was not reported officially until death (*Stiles* v. *John Nowlem and Co.*).[34] About the same time a miner's wife, the mother of four children, was awarded £600 compensation by the Newcastle County Court. Her husband had contracted a leptospiral infection while working underground in water. The company was not satisfied with

TABLE 5.—*Occupational Distribution*

10	swimmers
8	sewer workers
6	fish cutters
6	laborers
4	dairy workers
4	poultry dressers
4	who lived in cheap lodging houses
3	abattoir workers
2	laboratory workers
2	veterinarians
2	eating place operators
1	boseman on a fire boat
1	golfer who waded in a river
1	who had a boil on his forearm which broke while he was searching for a golf ball in dirty, stagnant water
1	butcher
1	who worked and slept in a rat infested kitchen
1	salesman in a meat and vegetable market
1	renovator in an old deserted carpet factory
1	quarry worker
1	machinist in a paper mill, whose wife first had the infection
1	garage mechanic
1	unemployed brick mason
1	who moved wet lumber and waded in a river
1	who lived in a rat infested home and frequently trapped rats
1	who lived on a city dump for 18 years
1	rat catcher
1	who waded in a sewer creek
1	kitchen worker
1	worker on the construction of a gas main
1	schoolgirl
1	housewife
2	occupations not known

the proof of the cause of death until necropsy, when the organisms were demonstrated in one of the kidneys.[35]

28. Bonnig, F. A.: Ein Fall von Weilscher Krankheit, verursacht durch einen Unfall, München. med. Wchnschr. **74**: 1628 (Sept. 23) 1927; abstr. J. A. M. A. **89**: 2231 (Dec. 24) 1927.

29. Hegler, C.: Weilsche Krankheit als Unfallfolge, Deutsche med. Wchnschr. **50**: 298 (Feb. 24) 1933; abstr. Trop. Dis. Bull. **30**: 370 (June) 1933.

30. Smith, H. E.: Correspondence, Brit. M. J. **1**: 39 (Jan. 5) 1935.

31. Rolleston, Humphry: Departmental Committee on Compensation for Industrial Diseases: Third Report to the Right Honourable the Secretary of State for the Home Department by the Departmental Committee Appointed to Inquire and Report as to Certain Proposed Extensions of the Schedule of Industrial Disease to Which Section 43 of the Workmen's Compensation Act, 1925, Applies, abstr. Foreign Letters (London), J. A. M. A. **107**: 1729 (Nov. 21) 1936, and Trop. Dis. Bull. **34**: 357 (April) 1937.

32. Compensation for Weil's Disease, Annotation; Brit. M. J. **2**: 722 (Oct. 10) 1936.

33. Compensation for Weil's Disease, Annotation; Brit. M. J. **1**: 1081 (May 25) 1935.

34. Medicolegal: Weil's Disease Contracted During Employment: Workmen's Compensation Case, Brit. M. J. **2**: 54 (July 4) 1936.

35. Medicolegal: Weil's Disease Again, Brit. M. J. **2**: 570 (Sept. 12) 1936.

In Australia, leptospiral infections were also made a compensable disease under the worker's compensation act. In 1936, 57 patients had received compensation, as shown in table 6.

In the United States the first case of leptospiral infection in which compensation was awarded was that of Glotzer.[36] The patient was a man aged 33 who was

TABLE 6.—*Disability Claimed in Australia for Leptospiral Infections*

Period of Incapacity	Number of Cases
1 to 2 weeks	7
2 to 3 weeks	15
3 to 4 weeks	15
4 to 5 weeks	9
5 to 7 weeks	2
7 to 8 weeks	4
10 weeks	1
12 weeks	1
24 weeks	1
Still receiving compensation after 26 weeks	1
Still receiving compensation after 36 weeks	1

engaged by a wholesale merchant in New York City to fillet fish. He became ill while at work on Oct. 2, 1937. His illness was that of a severe hemorrhagic jaundice, and the diagnosis of a leptospiral infection was established by serologic tests performed at the National Institute of Health. The case was first disallowed, when considered by the New York State Workman's Compensation Division. Following an appeal, however, an award for ten weeks of disability was made on June 2, 1938. Later, consideration was given to the possible causal relationship between the leptospiral infection and more recent symptoms suggesting a chronic cholecystitis, but to date no decision has been made.

Compensation was awarded in a second nonfatal case of leptospiral infection in a fish worker in New York on March 10, 1939.[37] The patient was a man aged 36 who became ill on Aug. 7, 1938. A blood specimen taken on the fifteenth day of illness was found to contain a high titer of leptospiral agglutinins.

In a third case of leptospiral infection compensation was also awarded in New York State.[38] A man aged 41, a street and sewer cleaner in Newark, fell from a truck on Sept. 22, 1938, injuring his right side. He was hospitalized for eight days because damage to the right kidney was suspected, but his clinical course was uneventful. Thereafter he convalesced in his two room shanty on the bank of the Erie Barge Canal until October 28. At that time he became acutely ill, suffered a hemorrhagic jaundice and died on November 10. The diagnosis of a leptospiral infection was confirmed by the examination of silver-stained sections of the lungs and kidneys. It was held that there was a causal relationship between the injury and the fatal infection. Subsequent independent investigations, however, point to an error in this connection, for thirty-six days elapsed between the accident and the onset of the fatal illness—a period greater than the recognized incubation period of leptospiral infections.

Realizing the potential hazards in certain occupations, employers have already undertaken preventive measures.[39] Attempts have been made to reduce the contamination of water and other materials by exterminating rats, eliminating refuse and rat proofing buildings. To destroy the leptospira organisms in nature, drainage of stagnant water and disinfection have been tried. An effort has been made to protect workers by the use of suitable clothing and by active immunization.

36. Glotzer, Solomon: Weil's Disease: Report of a Case in a Fish Worker, J. A. M. A. **110**: 2143 (June 25) 1938.
37. Farrell, E.: Weil's Disease—A Compensable Infection in New York State, New York State J. Med. **39**: 1969 (Oct. 15) 1939.
38. Kaufman, J. J.: Personal communication to the authors, 1940.
39. Bonnig,[29] Glotzer,[36] Taylor and Goyle.[32]

COMMENT

Leptospiral infection may be considered as an occupational hazard, provided the occupation exposes the patient to rats or dogs, or to moist materials contaminated by the urine of these animals, and provided that the illness follows such exposure by the recognized incubation period of from two to nineteen days. Certain other criteria should be fulfilled before a leptospiral infection is considered as compensable. A nonoccupational source of infection should be eliminated as a probability. If the patient has been swimming or wading in water that may have been contaminated by rats or dogs, it is more likely that this was the source of his infection. If the patient has had contact with a pet dog, the animal should be studied for evidence of a leptospiral infection. If the dog is proved to be a shedder of leptospira organisms that are pathogenic for guinea pigs, it is probable that infection arose from this source. A rat-infested residence must also be eliminated as a probable source of the infection. It is imperative to prove that the patient is suffering from a leptospiral infection and not from some other illness that may be easily confused with the leptospiral disease.

Of the laboratory methods available for the positive diagnosis of leptospiral infections, the most conclusive is the reproduction of the disease in guinea pigs that have been injected with macerated tissue or fluids from the patient, and recovery of leptospira organisms from these animals. Properly controlled serologic tests are reliable when the titer is found to be high or rising. An instance has been encountered in which a low agglutinin titer persisted throughout an illness that was confused clinically with a leptospiral infection but that was finally considered to be of other causation.[40] Histologic sections of the patient's tissues stained by silver technics are acceptable evidence, provided organisms that have the characteristic morphology and staining characteristics of leptospira organisms are found.

Unreliable laboratory technics include those in which darkfield microscopy is used. Confusing artefacts are common in body tissue and fluids, as pointed out repeatedly in the medical literature.[41] In spite of this knowledge, many cases have been reported in the United States in which the diagnosis of a leptospiral infection was based solely on the finding of "organisms resembling leptospira" by darkfield microscopy. Pseudospirochetes have been observed in tissues and fluids of persons in good health and in those of patients suffering from other diseases. Many specimens "swarming" with these artefacts have been injected into susceptible guinea pigs without producing leptospiral infection. Other specimens exhibiting no leptospiral forms by darkfield microscopy have proved to be

40. McKeon, J. A., and Brown, H. C.: An Unusual Case of Jaundice in a Miner, Brit. M. J. **2**: 174 (July 25) 1936.
41. Artefact Spirochetes, Current Comment, J. A. M. A. **74**: 1783 (June 26) 1920. Eberson, Frederick: Spirochetes Derived from Red Blood Corpuscles, Arch. Dermat. & Syph. **1**: 638 (June) 1920; abstr. J. A. M. A. **75**: 200 (July 17) 1920. Kato, J.: On Spirochaeta-like Bodies in the Blood, Juzenkwai Zasshi, April 1925, vol. 30; Japan M. World **5**: 358 (Dec. 15) 1925; abstr. Trop. Dis. Bull. **23**: 607 (Aug.) 1926. Koga, G., and Otsubo, I.: Spirochete-like Bodies in Cultures of Certain Bacteria, Kitasato Arch. Exper. Med. **2**: 207 (Dec.) 1919; abstr. J. A. M. A. **75**: 69 (July 3) 1920. Schultz, E. W.: The Pseudospirochetes Derived from Red Blood Cells, J. Lab. & Clin. Med. **8**: 375 (March) 1923. Thompson, J. G.: The Common Occurrence of Pseudospirochetes in the Blood of Blackwater Fever Cases and of Normal Human Beings and Animals When Using the Technic of Blanchard and Lefrou, J. Trop. Med. & Hyg. **26**: 251 (Aug. 1) 1923.

infectious for guinea pigs. Several American authors recently interviewed have expressed the opinion that they have confidence in their own darkfield technics yet are skeptical of the results of others. Others have admitted that they could not rely on their own darkfield observations and that they would not accept those of others.

Several cases have been studied carefully in which the clinical history or postmortem findings were compatible with a leptospiral infection, and yet no positive evidence of an infection, as ascertained by multiple animal injections, serologic tests or the study of histologic sections has been found. A diagnosis of leptospiral infection that is based solely on the clinical history, darkfield microscopy or gross postmortem findings is therefore unreliable.

SUMMARY

Certain occupations expose workers to a risk of leptospiral infection that is greater than the risk run in private life. Compensation for an industrial infection is therefore justified, provided nonoccupational circumstances can be ruled out as more likely sources of the infection and that the leptospiral etiology can be established conclusively.

Leptospiral infection should be considered as an occupational hazard—not as an occupational disease.

SPECIAL NOTE.—Since the present paper was accepted for publication, we have learned of more than a score of additional cases, details of which will be reported in a forthcoming paper on the incidence of leptospiral infections in the United States.

THE TREATMENT OF PEPTIC ULCER WITHOUT ALKALIS

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The use of alkaline substances in the management of peptic ulcer is almost universal, and such treatment is recommended in nearly all the current textbooks dealing with the subject.[1] It is commonly assumed that complete neutralization of gastric acidity represents the ideal condition for ulcer healing, although no proof of this assumption exists. Furthermore, it is not certain that the highly alkaline stomach content obtained periodically in the course of alkali therapy is conducive to healing. That the conventional methods of treatment are not entirely satisfactory is manifest by the fact that scarcely a year passes without one or more new "ulcer treatments" being advocated. At frequent intervals new kinds of neutralizing agents, supposedly with better virtues or fewer evils, are recommended.

The disadvantages of alkali therapy, which have been commonly recognized during its wide use, are:

1. Alkalis may produce secondary acid secretion. Crohn and Reiss [2] state that alkalis are second only to histamine in their power to stimulate acid secretion. Wosika and Emery [3] estimate that in the Sippy treatment fifty times as much alkali is given as would be required to neutralize all the acid normally secreted by the stomach, but even so the acidity is not entirely controlled. Two explanations are offered for this fact. The alkali may stimulate gastric secretion or it may induce more rapid emptying of the stomach. They express the belief that both factors are involved.

2. On occasion, alkalosis may be produced. This is particularly true of patients with pyloric obstruction, renal impairment or severe anemia. Eisele [4] has shown that all patients following the Sippy treatment suffer from some degree of chemical alkalosis, with an elevation of both the p_H and the carbon dioxide content of the plasma above normal. Although the patients who suffer from a full blown clinical alkalosis are relatively few, instances are numerous in which minor symptoms require the withdrawal of alkalis for a few days.

3. Some of the alkalis, notably calcium carbonate and phosphates and bismuth compounds, cause constipation. In most cases it is necessary to add magnesia as a cathartic, and in some cases an undesirable cathartic colitis develops.

4. As has been shown by Eisele,[5] the use of alkalis may result in the formation of kidney stones.

5. The use of aluminum hydroxide and aluminum sulfate, which recently has become quite widespread, may have deleterious effects. Therapeutic doses may interfere with the absorption of inorganic phosphates from the intestinal tract, thereby deranging the calcium metabolism of the body.[6] Constipation and fecal impaction may occur,[7] and even intestinal obstruction has been reported.[8]

For many years one of us (G. F. D.) has treated peptic ulcers essentially without the use of alkalis; that is, they were given only in individual doses when necessary to control pain early in the course of treatment. It is our purpose in this report to show not only that many patients recover from peptic ulcer without alkali therapy but also that it is doubtful whether the use of alkalis ever results in more rapid recovery. This idea is not new. Brinton [9] in 1862 said that many patients get well with diet alone, but this seems to have been largely forgotten.

EVALUATION OF EVIDENCE OF HEALING

The difficulty of evaluating any regimen of ulcer treatment is well recognized. Under a wide variety of treatments, or with no treatment at all, ulcer pain may disappear and the patient may be completely comfortable within a few days, yet roentgenologic and pathologic evidence may show an unhealed ulcer still present.

From the Department of Medicine, University of Chicago.

1. Brown, T. R., in Cecil, R. L.: A Textbook of Medicine, ed. 4, Philadelphia, W. B. Saunders Company, 1937. Easterman, G. B., and Balfour, D. C.: The Stomach and Duodenum, Philadelphia, W. B. Saunders Company, 1936. Hurst, A. F., and Stewart, M. J.: Gastric and Duodenal Ulcer, London, Oxford University Press, 1929. Osler, William: The Principles and Practice of Medicine, revised by H. A. Christian, ed. 13, New York, D. Appleton-Century Company, Inc., 1938. Palmer, W. L., in Portis, S. A.: Diseases of the Digestive System, Philadelphia, Lea & Febiger, 1941. Price, F. W.: A Textbook of the Practice of Medicine, ed. 5, London, Oxford University Press, 1937.

2. Cited by Hurst and Stewart.[1]

3. Wosika, P. H., and Emery, E. S.: The Effectiveness of the Sippy Regimen in Neutralizing the Gastric Juice of Patients if the Amount of Alkali Is Not Varied, Ann. Int. Med. **9**: 1070 (Feb.) 1936.

4. Eisele, C. W.: Changes in the Acid-Base Balance During Alkali Treatment for Peptic Ulcer, Arch. Int. Med. **62**: 1048 (June) 1939.

5. Eisele, C. W.: Role of Alkali Therapy for Peptic Ulcer in Formation of Urinary Calculi, J. A. M. A. **114**: 2363 (June 15) 1940.

6. Street, H. R., and Barlow, O. W.: The Relative Effects of Aluminum Hydroxide and Aluminum Sulfate on the Absorption of Dietary Phosphorus by the Rat, Am. J. Physiol. **133**: 465 (June) 1941. Freeman, Smith, and Freeman, Willie Mae: The Interference in the Absorption of Inorganic Phosphorus by Aluminum Hydroxide: Its Use in Children with Chronic Renal Insufficiency, Am. J. Physiol. **133**: 281 (June) 1941.

7. Kraemer, Manfred: The Use of Hydrated Magnesium Trisilicate in Peptic Ulcer, Am. J. Digest. Dis. **5**: 422 (Sept.) 1938. McIntosh, J. F., and Sutherland, C. G.: Use of Colloidal Aluminum Hydroxide in Treatment of Peptic Ulcer, Canad. M. A. J. **42**: 140 (Feb.) 1940.

8. Havens, W. P.: Intestinal Obstruction Caused by Colloidal Aluminum Hydroxide, J. A. M. A. **113**: 1364 (Oct. 21) 1939.

Gaither,[9] quoting Hurst and Eusterman, has reported numerous instances in which ulcers were "cured" by parenteral treatment, the patient being entirely symptom free but an ulcer as large as ever being found at operation.

The disappearance of occult blood from the stool is taken as an objective sign of healing, but this too is not

Fig. 1 (case 41).—Ulcer crater of the lesser curvature of the stomach in profile view. The crater measures 24 mm. in diameter and 9 mm. in depth. After forty-five days of treatment the crater had disappeared completely, leaving only a scar that was detected with difficulty.

wholly reliable. The disappearance of the gastric ulcer niche as detected by x-ray studies has for many years been accepted as the most satisfactory evidence of healing.[10] But, until Berg's controlled compression technic was introduced into this country in recent years, similar observations were not feasible for duodenal ulcers. Berg reported finding craters in 50 per cent of his cases of duodenal ulcer, but prior to the use of this technic craters in duodenal ulcers were seen in only the occasional case. Templeton, Marcovich and Heinz[11] state that "the best clinical evidence of activity and healing is the roentgenologic demonstration and the progressive and complete disappearance of the crater. Our observations confirm those of others that a large percentage of niches persist for a considerable time after symptoms are clinically relieved." These authors as well as others[12] warn that the disappearance of the crater is not necessarily synonymous with complete healing. The crater may be filled with granulation tissue but not yet epithelized. On occasion the crater may be filled with food, mucus or blood clot. Nevertheless, crater disappearance is the best clinical criterion of healing.

9. Gaither, E. H.: Therapy of Peptic Ulcer: Conservative Versus Radical, Am. J. Digest. Dis. & Nutrition **2**: 736 (Feb.) 1936.
10. Hamburger, W. W.: Roentgenological Studies in the Healing of Gastric and Duodenal Ulcers, Am. J. M. Sc. **155**: 204 (Feb.) 1918.
11. Templeton, F. E.; Marcovich, A. W., and Heinz, T. E.: Duodenal Ulcer: The Value of the Roentgenologic Demonstration of Crater, J. A. M. A. **111**: 1807 (Nov. 12) 1938.
12. Walko, K.: Concerning the Pain, the Latent Phase and Chronicity of Peptic Ulcer, Mitt. a. d. Grenzgeb. d. Med. u. Chir. **30**: 1, 1926.

METHODS AND MATERIAL

In the present study 41 patients with peptic ulcers were selected on the basis that their x-ray studies before treatment showed definite ulcer craters and that subsequent studies were made at sufficiently frequent intervals to indicate the course of the crater. As this was the only criterion for selection, this group should not show a more favorable response to treatment than the series as a whole. Actually, this selection probably affected the group adversely, for it included more than its share of severe cases requiring prolonged periods of hospitalization and but few completely ambulatory patients. This was true because the patients with long hospitalization afforded better opportunities for the repetition of x-ray studies at sufficiently short intervals. Six of the patients had suffered recent gross hemorrhages.

These patients were given 1 to 4 ounces (30 to 120 cc.) of a mixture of equal parts of milk and cream at hourly intervals during the day and often during the early evening. In a few instances in which cream was poorly tolerated, milk alone was given. When all distress was relieved, small amounts of bland foods were added gradually. Added vitamins were given in most cases. The importance of physical and mental rest was emphasized. Atropine was given in some cases. In 8 cases a few single doses of alkali were given to control pain. Attention was directed to the removal of foci of infection when such were present. Therapeutic aspiration of the stomach at bedtime, a common custom under many regimens, was not done. Instead, one or more bedtime aspirations were done when treatment was begun, and in cases showing residuum the quantity of milk and cream per hour was reduced until the stomach was practically empty at bedtime. We believe that the results so obtained are better

Fig. 2 (case 5).—Duodenal ulcer crater 23 mm. in diameter (shown to be 15 mm. deep in other views). After forty-eight days of treatment the crater had disappeared but definite bulb deformity remained. Subsequent x-ray studies confirmed the disappearance of the crater.

than giving an arbitrary amount of feedings and then aspirating the retained food every night. We further made an attempt to adjust the volume of the hourly feedings so as to induce the complete comfort of the patient.

Seven patients were suffering from gastric ulcers [13] and 34 from duodenal ulcers. Two of the latter group were followed through two distinct attacks, thus making 36 duodenal ulcers in the series. Twenty-six of the patients were men and 15 were women. The ages ranged from 23 to 74 years, the average age being 40 years. Five were entirely ambulatory and 36 were hospitalized for periods varying from three to fifty-two days, the average being twenty-four days.

RESULTS

The time of disappearance of the x-ray crater in each case is represented by a bar in the accompanying chart. The left end of the bar indicates the time (days of treatment) of the last x-ray examination at which a crater was demonstrated, and the right end the time of the first examination at which the crater had disappeared. In 25 cases the last demonstration of the crater was at the initial examination. It will be noted that in 17 cases an average of seventy-six days (ranging from fifty-two to one hundred forty days) elapsed between these two pertinent examinations. Unfortunately, this introduced a considerable latitude as to the actual time

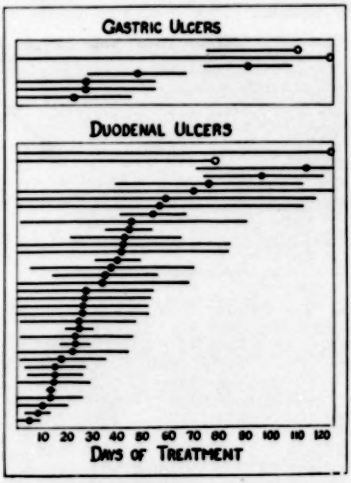

Fig. 3.—Time of x-ray crater disappearance. Each case is represented by a bar, the left end of which indicates the time of the last x-ray examination at which a crater was demonstrated; the right end indicates the first examination at which the crater had disappeared. The midpoint of each bar, indicated by a dot, represents the most probable time of crater disappearance. A circle at the right end of a bar indicates that the crater was still present at the last examination.

of crater disappearance. The midpoint of each bar, indicated by a dot, represents the most probable time of disappearance (the mean time of crater disappearance).

13. In 1 case, two craters were followed to healing gastroscopically instead of roentgenologically.

The table summarizes the time of the disappearance of the craters in the cases of duodenal ulcer. It will be noted that 86 per cent had disappeared within seventy-five days (mean time of crater disappearance) and that in 70 per cent of the cases the crater was actually demonstrated to have disappeared within seventy-five days. In 2 cases (6 per cent) the craters did not disappear completely although they decreased in size considerably. One of the patients was symptomatically well and apparently in good health after the first week of treatment. In the second case a duodenal ulcer crater disappeared promptly without the use of alkalis one year before, but in the attack under consideration the crater persisted for seventy-five days, although it became shallow with indistinct margins, thus showing definite evidence of healing. Subsequently the crater disappeared with the administration of alkalis.

Disappearance of X-Ray Crater in Duodenal Ulcers

Days of Treatment	Examination When Crater Had First Disappeared: No. of Cases (Cumulative)	Examination When Crater Had First Disappeared: Per Cent	Mean Time of Crater Disappearance: No. of Cases (Cumulative)	Mean Time of Crater Disappearance: Per Cent
Within 15 days	3	5	8	22
Within 30 days	10	28	18	50
Within 45 days	13	36	27	75
Within 60 days	21	58	30	83
Within 75 days	25	70	31	86
More than 75 days	34	94	34	94
Nonhealing	2	6	2	6

The cases of gastric ulcer were too few for adequate analysis, but in general they followed a similar course. Again, in 2 instances healing did not take place. In 1, surgical treatment was necessary after one hundred and ten days. The other patient left the hospital against advice after three days of treatment and he followed management very poorly. His ulcer distress had been present for over twenty years.

No definite correlation was found between the age of the patient and the time of crater disappearance or between the duration of ulcer history and crater disappearance.

As others have noted, complete relief of symptoms occurred in nearly all cases long before the crater disappeared. In 31 cases all symptoms were controlled within one week. In 9 cases the progress notes concerning relief from pain were inadequate. In the case in which the duodenal ulcer did not disappear during the second attack, symptoms persisted irregularly for many weeks.

The benzidine reaction of the stools was negative in 14 cases despite the presence of a crater. In 12 cases a positive test became negative before the x-ray examination which first showed the disappearance of the crater. In 1 case a positive benzidine test persisted after the crater disappeared.

COMMENT

These data demonstrate that healing of peptic ulcers will progress satisfactorily without the attempted neutralization of gastric acidity with alkalis. It appears that it is only in the exceptional case that recovery will take place on alkali therapy and not without it. On the other hand, we have observed several patients who

did poorly on Sippy treatment whose response was notably better with the management described in this paper.

The speed of recovery compares favorably with that under any other type of treatment. We were unable to find in the literature a series of duodenal ulcers treated by the Sippy regimen and adequately observed by x-ray studies of crater behavior. However, the results in our series of duodenal ulcer compare very favorably with those obtained by Jordan and Boynton [14] in their series of 41 cases of gastric ulcer treated by Sippy management—although several authors [15] have stated the belief that gastric ulcers heal more rapidly than duodenal ulcers. In their series of 41 patients with ulcers of the lesser curvature, 9 were operated on because of inadequate diminution of crater or persistence of symptoms, 13 healed, with crater disappearance, within twenty-one to twenty-eight days of hospitalization, and 19 healed within four to ten weeks after leaving the hospital.

Our results with milk and cream without alkalis are not surprising in view of the observations of Wosika and Emery.[3] They performed half hour titrations of free and total acid of the gastric contents of ulcer patients receiving hourly feedings of milk and cream, and subsequently of the same patients receiving the first day Sippy treatment (hourly milk and cream plus hourly powders on the half hour). On the Sippy treatment the free acid was depressed an average of only 2 clinical units in comparison with the titrations with milk and cream alone. The patients were then divided into four groups according to the effectiveness of acid control, and no appreciable advantage of the first day Sippy treatment over the treatment with milk and cream was noted.

The treatment of peptic ulcer is usually considered to consist of two phases, the treatment of the immediate attack and the prevention of recurrences. Peptic ulcer is notoriously a chronic disease subject to recurrences, perhaps spontaneously, perhaps under adverse conditions such as fatigue, emotional stress and infections. It is not within the scope of this paper to discuss the prevention of recurrences. It is illogical, however, to expect the use of alkalis to prevent recurrences unless one is willing to advise such use throughout the life of the patient.

In many instances such ingestion of alkali would probably be more detrimental than the ulcer itself. Except for general hygienic measures, adequate prevention of recurrences awaits the solution of the problem of the etiology of ulcer.

SUMMARY

Under hourly treatment with milk and cream without the use of alkalis, the speed of healing of gastric and duodenal ulcers compares favorably with that under other methods of treatment. Such treatment of a series of 41 patients brought about (1) the prompt disappearance of symptoms and the complete comfort of the patient, (2) the disappearance of occult blood from the stool when it was present and (3) the disappearance of the x-ray crater.

14. Jordan, S. M., and Boynton, L. C.: Treatment of Gastric Ulcer, Tr. Am. Therap. Soc. **34**: 56, 1934.
15. Clark, D. M., and Geyman, M. J.: Roentgen Evidence of Healing in Duodenal Ulcer, J. A. M. A. **102**: 107 (Jan. 13) 1934. Delario, A. J.: A Roentgenologic Follow-up of One Hundred and Twenty-Five Cases of Peptic Ulcer, with Clinical and Laboratory Findings, Am. J. Roentgenol. **34**: 190 (Aug.) 1935.

Clinical Notes, Suggestions and New Instruments

ALOPECIA FROM CYVERINE HYDROCHLORIDE

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Our purpose in this paper is to report the occurrence of alopecia following the use of a new antispasmodic drug. In this case, the defluvium followed the daily ingestion, for thirty days, of cyverine hydrochloride (Stearns) and rapidly progressed to involve the entire scalp.

REPORT OF CASE

History.—A white woman aged 49 was seen in consultation on June 6, 1941, complaining of an extreme degree of falling out of the hair of ten days' duration. She gave a history of spasmodic pain in the lower part of the abdomen for the previous three months, for which she had been under the care of her family physician. During this period, several examinations including roentgen ray and proctologic studies

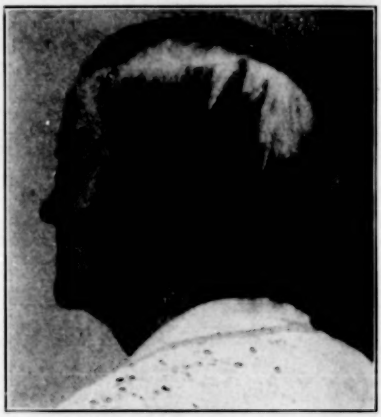

Fig. 1.—Appearance of scalp on June 23, showing loss of hair from the temporo-occipital region.

of the intestinal tract disclosed no abnormalities, and the diagnosis of a functional intestinal disorder was made. On April 24 the patient was instructed to take cyverine hydrochloride (Stearns), an antispasmodic, for the relief of her symptoms.

She took 40 mg. daily for three days and then 80 mg. daily for four days. At that time, she complained of a sensation of numbness of the arms. She continued to take the pills until May 9, after which she stopped taking the medication because she had finished her prescribed supply (800 mg.). On May 18 she again took 40 mg. daily, but within three days the patient noticed that ordinary combing of the hair resulted in the accumulation of hundreds of hairs on the comb. This defluvium increased so rapidly that within ten days the occipital region of the scalp was almost entirely devoid of hair. At the same time the patient noticed that her skin was extremely dry and was covered with fine, branny scales. She also observed that her eyebrows and eyelashes were turning white.

The patient also gave a history of having had her hair dyed several times during the past three years. The last application of dye was made two weeks prior to the occurrence of alopecia.

The same dyes were employed at that time as during the three year period. The patient did not experience any local reactions on the scalp at any time following the application of these dyes.

Examination of the skin at this time showed a loss of axillary hair in addition to the diffuse alopecia of the scalp. The skin of the entire body showed a mild type of exfoliative dermatitis

Fig. 2.—Appearance of occipital area on June 23.

and was beginning to peel in small flakes over the forearms and legs. On questioning, the patient stated that she had perspired so infrequently in the previous two weeks as to render unnecessary the use of a proprietary antiperspirant preparation. Prior to this episode the antiperspirant preparation had to be applied daily.

Laboratory Examinations.—Urinalysis consistently showed the urine to be normal. The hemoglobin of the blood was 12.6 Gm., the erythrocyte count was 4,600,000 and the leukocyte count was 6,200 with 58 per cent neutrophils, 34 per cent lymphocytes, 6 per cent monocytes and 2 per cent eosinophils. The blood Wassermann and Kahn reactions were negative. Patch tests performed on the back with the dyes which had been applied to the patient's scalp gave positive reactions (La Rue dye). The basal metabolic rate was within normal limits.

Course.—The patient is still under observation and treatment. The falling out of the hair has been continuous and progressive. At present there is very little hair left on the scalp and in the axillas, and the eyelashes and eyebrows are almost white.

COMMENT

The definition of alopecia states that it is a partial or total loss of hair. When it occurs in patches, it is designated alopecia areata.

The etiology of alopecia is unknown in the majority of cases. Controversy and speculation is still rampant concerning the cause or causes of such conditions as premature alopecia, seborrheic alopecia and alopecia areata. However, certain definite etiologic factors have been recognized. It is known that alopecia may follow the high temperatures which occur in pneumonia, influenza and typhoid. It is also known that certain endocrine changes may lead to alopecia, as in myxedema, cretinism and acromegaly. Alopecia may occur from local disease, especially following burns, furunculosis, ulcerating syphilids and cicatrizing cutaneous diseases (pseudopelade, folliculitis decalvans). When alopecia occurs from local causes, it is always due to destruction of the hair follicles by ulceration and by the formation of scar tissue. Thus it is conceivable that alopecia might follow the application of strong dyes to the scalp, although there have been no reports of such an occurrence in the literature. However, in such an instance the alopecia would be preceded by inflammation, destruction of the hair follicles by ulceration and by the formation of scar tissue. None of these processes occurred in this case and the scalp was normal in every respect except for the absence of hair. A dye reaction would also lead to brittleness of the hair, so that there would be many short stumps present in the thinned areas. In this case, the hairs were of normal texture and definitely not brittle. When a moderate degree of traction was exerted on the hair shaft it slid out of its follicle very easily but did not break any more readily than normal hair.

Alopecia from drugs, although uncommon, has been reported several times. The use of gold salts in the treatment of arthritis resulted in alopecia in 2 reported cases,[1] and in 1 of these [2] the loss of hair on the scalp was total in extent and was not followed by regrowth. The use of thallium salts for epilation has been practiced for a period of more than ten years in the treatment of tinea capitis.[3] The thallium salts are not unattended by toxic symptoms and the drug most be used judiciously. Alopecia areata has been artificially produced by the intravenous injection of quinine hydrochloride and

Fig. 3.—Appearance of right axilla on June 23, showing loss of hair.

ethyl carbamate.[4] The so-called toxic theory, which states that alopecia areata is due to the action of certain toxins on the autonomic nervous system and on the hair itself, was

1. Belinfante, A. J. G.: Change in Nails and Alopecia After Gold Dermatitis, Nederl. tijdschr. v. geneesk. **81**: 2613-2614 (June 5) 1937. Bertier and Bocquillon.[2]

2. Bertier, L., and Bocquillon, P.: Total Persistent Alopecia Following Gold Sodium Thiopropanol Sulfonate Therapy for Arthritis, Bull. Soc. franç. de dermat. et syph. **39**: 1335 (Nov.) 1932.

3. Pardo-Castello, V.; Mestre, J. J., and Rio, Enrique: Tinea of the Scalp: Treatment with Thallium Salts, Arch. Dermat. & Syph. **19**: 409 (March) 1929.

4. Bergman, Alexander: Alopecia Areata, Artificially Produced by Intravenous Injection of Quinine Hydrochloride and Ethyl Carbamate, Arch. Dermat. & Syph. **35**: 285 (Feb.) 1937.

substantiated in some measure by the finding of arsenic and lead in the body fluids and tissues in a series of these cases.[5] The presence of these heavy metals in the body was accounted for by the widespread prevalence of arsenic and lead in foods, drugs and liquids due to contamination and spray residue. Chronic arsenical poisoning has long been recognized as a causal agent in alopecia and precancerous keratoses.

In this case, the drug which produced the alopecia is known as cyverine hydrochloride (Stearns). The chemical formula is methyl-bis-beta-cyclohexyl-ethyl-amine hydrochloride. It is a white crystalline powder, soluble in water up to 1 per cent and freely soluble in alcohol. It has a bitter taste, and on mucous membranes it has a mild local anesthetic effect. The empirical formula is $C_{17}H_{33}N.HCl$.

The structural formula of the cyverine base closely resembles that of papaverine. The structural formulas of cyverine and papaverine differ mainly by a break in the isoquinoline ring in cyverine, by which a prolongation in the chain is produced, each end of the chain carrying a cyclohexyl ring.

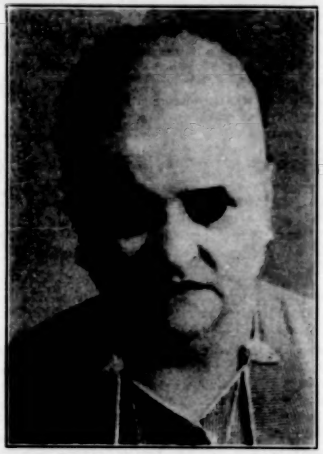

Fig. 4.—Appearance of scalp on July 14, showing almost total loss of hair.

Pharmacologic studies have shown that cyverine hydrochloride acts directly on smooth muscle tissue because it produced complete relaxation of smooth muscle sections (small intestine of rabbit) when the latter were (1) contracted by such parasympathetic stimulants as pilocarpine and arecaline, (2) contracted by a smooth muscle stimulant such as barium chloride, (3) atropinized and contracted by barium chloride and (4) nicotinized and contracted by barium chloride.

Clinical studies of 31 cases[6] reported but not cited by the manufacturer showed that cyverine hydrochloride has a powerful antispasmodic effect. It is apparently of value in disorders such as pylorospasm and other spastic conditions affecting the gastrointestinal tract. In this series of cases no toxic symptoms were reported other than nausea, "heartburn" and slight dryness of the throat.

The suggestion is advanced that the effects of the drug on smooth muscle caused complete relaxation of the erector pili muscles around the hair follicles. In this manner loosening of the hair shaft and subsequent alopecia may have occurred.

5. Myers, C. N.; Throne, Binford, and Kingsbury, J.: Toxic Action of Metals in Alopecia Areata, New York State J. Med. **33**:991 (Aug. 15) 1933.

6. Form 2625, a pamphlet entitled "Cyverine Hydrochloride," issued by Frederick Stearns & Co., Detroit.

SUMMARY

1. A white woman aged 49 years ingested 920 mg. of cyverine hydrochloride (Stearns), an antispasmodic drug, within a period of thirty days. At the end of this time alopecia of the scalp developed.

2. The alopecia was progressive and resulted in approximately complete loss of hair from the scalp.

Fig. 5.—Another view of patient on July 14.

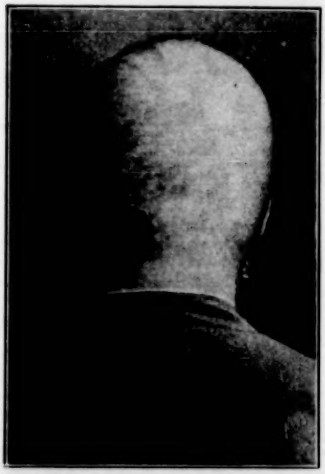

Fig. 6.—Another view of patient on July 14.

3. The eyebrows and eyelashes turned a grayish white shortly after the occurrence of alopecia.

4. There was an associated exfoliative dermatitis of the skin and a diminution in perspiration.

2 East Fifty-Fourth Street.

HYPERTENSION IN A PATIENT WITH BILATERAL RENAL INFARCTION

CLINICAL CONFIRMATION OF EXPERIMENTS IN ANIMALS

MYRON PRINZMETAL, M.D.; NATHAN HIATT, M.D., AND L. J. TRAGERMAN, M.D., LOS ANGELES

Goldblatt,[1] in a series of historic experiments, showed that hypertension was induced in animals by constricting the renal arteries. Numerous investigators have confirmed and amplified these observations on various animals, and it is now universally admitted that lasting hypertension follows the experimental induction of partial renal ischemia.

Goldblatt [2] later showed that hypertension also occurred following complete occlusion of one renal artery provided the renal vein was not obstructed.

Taquini,[3] modifying the Goldblatt procedure, showed that a temporary rise in blood pressure occurred in dogs on the reestablishment of the circulation in kidneys rendered completely ischemic for a period of from four to six hours. Taquini's observations have been confirmed by us on cats, dogs, rats and guinea pigs,[4] although negative results were obtained in

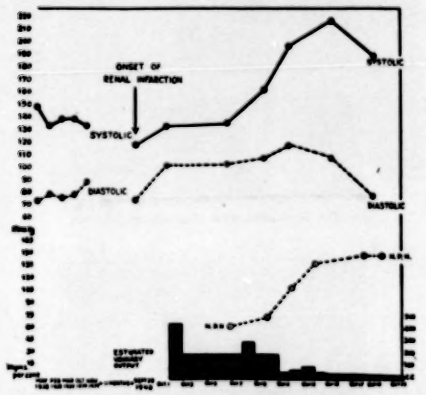

Fig. 1.—The effect of renal infarction on blood pressure, nonprotein nitrogen and urinary output. Note rise in blood pressure and nonprotein nitrogen; also decrease in the estimated urinary output.

rabbits.[5] The mechanism responsible for the form of hypertension induced by the Taquini method has been the subject of extensive investigation. It was found that an extract of a kidney made completely ischemic by clamping the renal pedicle for from four to six hours contained more pressor substance (renin) than that prepared from the opposite kidney with the circulation unimpeded.[6] Later it was shown that the same pressor substance or a similar one was present in perfusates of completely ischemic kidneys but absent from perfusates of normal kidneys as well as from perfusates of ischemic tissues other than kidney. It was conclusively proved that the pressor substance found in perfusates of completely ischemic kidneys is the cause of this type of hypertension,[7] and there was strong evidence to show that this pressor material is renin or a closely related substance.

The sequence of events during complete renal ischemia and following the reestablishment of the renal circulation can be reconstructed as follows: As a result of complete renal ischemia more renin is formed in the kidney, or that which is present becomes more readily available for extraction. Normally the kidney secretes little or no renin, since perfusates of normal kidneys do not have pressor properties. When the kidney is deprived of its blood supply, pressor material passes from the depots of manufacture and storage through the vascular endothelial cells into the renal blood vessels. A small amount of the pressor substance reacts with some of the plasma in these vessels to form a minute amount of heat-stable pressor substance,[8] but the bulk of transported material remains unaltered during the period of ischemia. When the renal blood supply is restored, the pressor substance enters the general circulation and a rise of blood pressure takes place, presumably through its transformation into a heat-stable substance termed angiotonin [9] or hypertensin.[10]

In this communication we wish to report confirmation in a clinical case of some of the aforementioned observations on experimental animals. A patient suffering from chronic rheumatic heart disease was observed to become hypertensive following complete occlusion of both main renal arteries, the blood pressure having been followed from normal to hypertensive levels. Renal perfusates prepared immediately after death revealed the presence of appreciable quantities of a pressor substance presumably similar to that which causes the hypertension following complete renal ischemia in animals.

REPORT OF CASE

History.—A woman aged 53 was admitted to the Cedars of Lebanon Hospital on Sept. 1, 1940. On the previous evening severe epigastric pain suddenly developed and she became faint. Within the next few minutes she became nauseated and dyspneic. Because of the persistence of these symptoms she was admitted to the hospital. She was known to have had rheumatic heart disease of many years' standing. Her blood pressure taken on several occasions before the present episode was found to be normal.

Examination.—On admission to the hospital the radial pulse was 120 a minute, with evidence of auricular fibrillation. There was no cardiac enlargement and the blood pressure was 110 systolic and 95 diastolic.

Course.—The day after admission her blood pressure was 130 systolic and 100 diastolic and she was given quinidine, which converted the auricular fibrillation to normal rhythm. On the tenth day the blood pressure was 170 systolic and 110 diastolic and on the twelfth day 200 and 120. The urinary secretion, which had been diminishing progressively, almost ceased. On the eighteenth day, the blood pressure was 190 systolic and 100 diastolic and the specific gravity of the urine

From the Cedars of Lebanon Hospital and the University of Southern California School of Medicine.

Endowed by grants from the Darian Foundation for Medical Research and the Beaumont Trust Fund.

1. Goldblatt, Harry; Lynch, James; Hanzal, R. F., and Summerville, W. W.: Studies on Experimental Hypertension; Production of Persistent Elevation of Systolic Blood Pressure by Means of Renal Ischemia, J. Exper. Med. **59**: 347-379 (March) 1934.

2. Goldblatt, Harry: Studies on Experimental Hypertension; Pathogenesis of Experimental Hypertension Due to Renal Ischemia, Ann. Int. Med. **11**: 69-103 (July) 1937.

3. Taquini, A. C.: Liberación de substancia hipertensora en el riñón completamente isquemiado, Rev. Soc. argent. de biol. **14**: 422-428 (Oct.) 1938; Production of Pressor Substance by Totally Ischemic Kidney, Am. Heart J. **19**: 513-518 (May) 1940.

4. Lewis, H. A.; Leo, S. D., and Prinzmetal, Myron: The Effect of Reestablishment of Circulation in Completely Ischemic Kidneys upon the Blood Pressure of Cats, Dogs, and Rats, Am. Heart J. **21**: 319-325 (March) 1941.

5. Prinzmetal, Myron; Lewis, H. A.; Taggart, John; Wilkins, Howard, and Drury, D. R.: The Effect of Transplanted Ischemic Kidneys and of Temporary Complete Renal Ischemia upon the Blood Pressure of Rabbits, Am. Heart J. **20**: 525-538 (Nov.) 1940.

6. Leo, S. D.; Prinzmetal, Myron, and Lewis, H. A.: Observations upon the Pressor Substance Causing the Rise in Blood Pressure Following the Termination of Temporary, Complete Renal Ischemia, Am. J. Physiol. **131**: 18-26 (Nov.) 1940.

7. Prinzmetal, Myron; Lewis, H. A., and Leo, S. D.: Etiology of Hypertension Due to Complete Renal Ischemia, Proc. Soc. Exper. Biol. & Med. **43**: 696-699 (April) 1940; Etiology of Hypertension Due to Complete, Renal Ischemia, J. Exper. Med. **72**: 763-776 (Dec.) 1940.

8. Page and Helmer.[9] Braun Menendez, Fasciolo, Leloir and Munoz.[10]

9. Page, I. H., and Helmer, O. M.: Crystalline Pressor Substance (Angiotonin) Resulting from Reaction Between Renin and Renin Activator, J. Exper. Med. **71**: 29-42 (Jan.) 1940.

10. Braun Menendez, E.; Fasciolo, J. C.; Leloir, L. F., and Munoz, J. M.: La substancia hipertensora de la sangre del riñón isquemiado, Rev. Soc. argent. de biol. **15**: 420-425 (Nov.) 1939.

had fallen to 1.008. Measurable quantities of albumin were present in the urine, and in the sediment there were numerous leukocytes, epithelial cells and a moderate number of erythrocytes but no casts. The nonprotein nitrogen rose to 72 mg. per hundred cubic centimeters on the seventh day after admission to 148 mg. on the day before death. There was increasing

Fig. 2.—Artist's drawing showing complete thrombotic occlusion of right renal artery (*a*) and extension of thrombus into left renal artery (*b*). Note multiple infarcts of kidney. A portion of thrombus lower down in the aorta is shown.

stupor, and death occurred on September 21, three weeks after admission (fig. 1).

A clinical diagnosis of occlusion of both renal arteries was made. The most probable cause was considered to be embolism, although the possibility of a dissecting aneurysm of the aorta involving the orifices of both renal arteries was also considered.

Postmortem Examination.—Autopsy was performed within one hour after death. The heart weighed 275 Gm. All chambers except the left ventricle were dilated and there were no mural thrombi. Moderate fibrosis with stenosis of the mitral valve was present, as well as slight thickening of the aortic valve. The aorta showed severe atheromatous disease with ulceration of the intima. An irregular friable gray-brown thrombus 3.5 cm. in length incompletely obstructed the aorta at the level of the renal arteries. The entire length of the major right renal artery and the first portion of the left renal artery were occupied by similar thrombotic material (fig. 2). A separate inferior renal artery on the right side was patent except where it was obstructed at its orifice by thrombus. The intima of the aorta and renal arteries underlying the thrombi showed atheromatous changes similar to those found elsewhere. Another more recent thrombus overlay the mouth of the inferior mesenteric artery and partially saddled the bifurcation of the aorta.

The left kidney weighed about 175 Gm. The capsule was adherent and thickened. Except for the lower pole, which

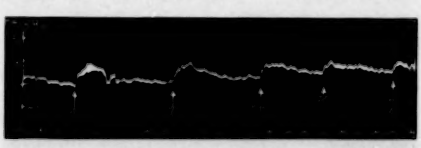

Fig. 3.—Effect of intravenous injection of perfusate from infarcted kidney on blood pressure of cat anesthetized with soluble pentobarbital. At the first arrow 2 cc. of perfusate was injected and at the second, third and fourth arrows the injections were 4 cc. Note diminishing pressor effect (tachyphylaxis). Time in minutes.

appeared fairly normal on gross examination the remainder of the kidney was mottled by numerous confluent zones of recent infarction.

The right kidney weighed 190 Gm. The renal artery was patent beyond the occlusion in its first portion and showed only slight narrowing by intimal atherosclerotic change. The capsule was adherent and recent infarction involved almost the entire kidney, both cortex and medulla, except for an ill defined zone at the upper pole. A large tributary of the renal vein was obstructed by recent antemortem thrombus. The lower urinary tract was normal. The spleen weighed 200 Gm. and contained numerous infarcts.

Histologic Examination.—The renal arteries and aorta in the region of the major thrombus showed varying degrees of intimal atheromatous changes but no inflammatory lesions. The oldest portions of the thrombus were found in the medium size branches of the left renal artery within the kidney, where organization was well established. In the left renal artery the process appeared more recent than within the kidney but older than in the aorta and right renal artery. The renal parenchyma on both sides showed the typical changes of infarction. In the portions of both kidneys which were grossly spared, the histologic appearance varied from normal to intense hyperemia and tubular degeneration.

The anatomic diagnosis was chronic rheumatic disease of the mitral and aortic valves with mitral stenosis; occlusion by thrombus formation, probably embolic in origin, of the left renal artery with extension to the aorta and right renal artery; extensive bilateral renal infarction; multiple infarcts of the spleen; severe atherosclerosis of the aorta and coronary arteries, and thrombus near the bifurcation of the aorta.

DEMONSTRATION OF PRESSOR SUBSTANCE IN PERFUSATES OF PATIENT'S KIDNEY

Since the sequence of events in this case appeared to follow the pattern of experimentally induced hypertension, it was resolved to prove, if possible, whether the mechanism for the

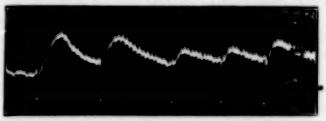

Fig. 4.—Effect of experimentally prepared renal pressor substance on the blood pressure of a cat anesthetized with soluble pentobarbital. Note similarity to results shown in figure 3.

production of hypertension in man is the same as in experimental animals. Accordingly, when the patient was moribund, permission for autopsy was obtained and preparations were instituted for making and testing a perfusate of one of her kidneys as soon as possible following death. When it was apparent that the obstructing thrombus (with resultant infarction) extended only a short distance into the renal artery on the right side, this vessel was cannulated beyond the point of obstruction and the kidney removed for perfusion. The experiment was performed twenty minutes after death.

Procedure.—Fifty cc. of warm physiologic solution of sodium chloride was reperfused through the kidney five times. Twenty cc. of a bloody perfusate was obtained. This was tested by intravenous injection into a cat anesthetized with soluble pentobarbital, the blood pressure being recorded from the carotid artery in the usual manner.

Results.—Two cc. of perfusate had a prolonged pressor effect. The test animal was then given repeated injections of 4 cc. of the perfusate, with the result that rapidly decreasing pressor responses were obtained (fig. 3). Another cat was then given 5 mg. of cocaine, followed by another injection of the perfusate. There was no change in pressor response, proving that the pressor substance was not parahydroxyphenylethylamine.[11] The shape of the pressor curve and the duration of the pressor response were similar to those obtained by intravenously injected

11. Tainter, M. L., and Chang, D. K.: Antagonism of Pressor Action of Tyramine by Cocaine, **30**: 193-207 (Jan.) 1927.

perfusate of completely ischemic kidneys of cats. Previous to this study we had tested perfusates from 4 fresh human kidneys, 2 of which were obtained immediately after death at autopsy and 2 following surgical removal. None of these perfusates which were prepared and tested in precisely the same manner as described exhibited a pressor effect.

COMMENT

The pressor substance found in perfusates of the ischemic kidney of the patient was similar to that present in perfusates of ischemic kidneys of animals because:

1. The pressor curves in the two were of similar configuration.

2. Both substances displayed the property of tachyphylaxis.

3. The pressor response was not altered by a previous injection of cocaine in either instance, excluding parahydroxyphenylethylamine as the cause for the hypertension.

4. Negative evidence is supplied by the fact that the pressor substance was not found in perfusates of nonischemic kidneys of either human beings or cats.

It will be recalled that when the patient was admitted to the hospital the blood pressure was normal and rose within the space of a few days to hypertensive levels and remained so until death. The postmortem examination not only confirmed the clinical impression of occlusion of both renal arteries but also excluded other possible causes for the sudden development of hypertension. It would appear, therefore, that this case furnishes a perfect example of hypertension following relatively acute renal ischemia in a human being. Since it has been proved that the pressor substance detected in perfusates of completely ischemic kidneys of cats is responsible for the postischemic hypertension in the Taquini experiments,[7] the finding of the same pressor substance in perfusates of the patient's ischemic kidney strongly suggests that the mechanism responsible for the patient's hypertension was the same as that which obtains in the experimental animals. Whether or not this substance is responsible for other types of clinical hypertension remains for future studies to decide.

From the clinical point of view this case is also of interest because it serves as a reminder that renal infarction may be followed by hypertension. Statements are often made that hypertension does not occur under these circumstances; extremely few articles state that an elevation of blood pressure may follow renal infarction.[12]

SUMMARY

A patient suffering from chronic rheumatic heart disease became hypertensive within the space of a few days following an acute attack of severe abdominal pain. The elevation of blood pressure was accompanied by increasing suppression of urine short of complete anuria and by progressive nitrogen retention, and death occurred in uremia. At the postmortem examination there were found occlusion of both main renal arteries by thrombus formation, probably of embolic origin, and almost complete infarction of both kidneys. The train of events being identical with those occurring in the experimental production of hypertension, this case offers a perfect example in a human being of hypertension resulting from acute renal ischemia.

Perfusates that were prepared immediately after death from one of the intarcted kidneys revealed the presence of a pressor substance which is presumably the same as that responsible for the hypertension that follows the termination of complete renal ischemia in experiments on animals. Attention is called to hypertension as a neglected sign in occasional cases of renal infarction.

4833 Fountain Avenue.

12. Fishberg, Arthur M.: Hypertension and Nephritis, ed. 4, Philadelphia, Lea & Febiger, 1939, New York.

Special Article

FIFTH ANNUAL SUMMARY OF FOURTH OF JULY INJURIES

DUE TO FIREWORKS AND EXPLOSIVES

SECOND SERIES

In 1937 the American Medical Association resumed its annual summaries of injuries resulting from the celebration of the Fourth of July with fireworks. This report for 1941 is the fifth of the reports in the series. The second series constitutes a resumption of similar summaries which were published from 1903 to 1916 and discontinued thereafter because of the reduction in the number of such accidents. Unfortunately the great increase in fireworks injuries again made this problem a matter of serious public importance.

DEATHS

Eleven deaths were reported in 1941 as directly due to the celebration of the Fourth of July with fireworks or other explosives. The distribution by states in comparison with the figures for recent years is given in table 1. For the first time since 1937 the decline in total number of deaths has been reversed. Eleven deaths were recorded in 1941 as compared with eight in 1940. It is also noteworthy that for the first time none of the fatalities were due to burns resulting from the igniting of clothing from fireworks. Three deaths occurred in Chicago, one from burns due to explosion of firecrackers, one in a small girl of 4 who bit into a torpedo and sustained extensive lacerations of the

TABLE 1.—*Deaths by State*

State	1941	1940	1939	1938	1937
California	0	1	2	0	1
District of Columbia	0	1	0	0	0
Connecticut	0	0	0	1	0
Florida	0	0	1	0	1
Idaho	0	0	0	0	6
Illinois	3	0	1	1	0
Indiana	1	0	1	3	0
Iowa	0	1	0	0	0
Maine	0	2	0	0	0
Maryland	0	0	1	2	1
Massachusetts	1	0	2	0	2
Mississippi	0	0	0	1	0
Missouri	1	0	0	0	0
New Jersey	0	1	1	0	1
New Mexico	1	0	0	0	0
New York	0	1	3	2	3
Ohio	1	0	0	1	1
Pennsylvania	0	0	0	6	0
Rhode Island	0	0	0	0	1
Tennessee	2	0	0	0	0
Texas	0	0	1	0	1
Utah	0	0	0	0	2
West Virginia	0	0	0	1	0
Wisconsin	1	0	0	0	0
Wyoming	0	1	0	0	0
Totals	11	8	13	18	20

TABLE 2.—*Total Injuries by Type*

Injuries	1941	1940	1939	1938	1937
Burns and lacerations	1,818	4,140	5,305	7,458	6,858
Loss of vision of one or both eyes	9	15	19	43	16
Injury to eye	104	109	158	281	294
Loss of finger, hand or other member	17	38	41	80	37
Internal injury, fracture or other serious accident	91	70	37	71	*
	2,039	4,402	5,560	7,933	7,205

* Not classified.

face, and the third in a visitor from Iowa who received a bullet wound from a gun used in noise making activities. Two boys in Memphis, Tenn., died as the result of injuries sustained when another boy tossed a lighted firecracker into the counter of fireworks in a shed from which the boys were selling. The other deaths resulted from a variety of accidents, including the premature explosion of a bomb in a fireworks display, a misdirected aerial bomb in another fireworks display, a gunshot wound, and an explosion which resulted while a bomb was being mixed.

TABLE 3.—*Injuries, by Type, Caused by the Celebration of the Fourth of July with Fireworks and Other Explosives*

	1941									
	Burns and Lacerations	Loss of Vision of One or Both Eyes	Injury to Eye	Loss of Finger, Hand or Other Member	Internal Injury, Fracture or Other Serious Accident	Total Injuries	Total Injuries, 1940	Total Injuries, 1939	Total Injuries, 1938	Total Injuries, 1937
Alabama	2	0	0	0	0	2	25	17	15	7
Arizona	1	0	0	0	0	1	12	30	16	22
Arkansas	1	0	1	0	0	2	5	3	2	7
California	200	2	8	1	20	231	251	650	509	485
Colorado	12	0	0	0	0	12	12	10	21	119
Connecticut	73	0	3	1	4	81	48	201	125	104
Dist. Columbia	63	0	5	0	2	70	68	57	51	78
Delaware	0	0	0	0	0	0	9	22	20	25
Florida	31	0	0	0	2	33	45	62	29	21
Georgia	1	0	0	0	0	1	6	5	7	9
Idaho	1	0	0	0	0	1	7	7	5	52
Illinois	187	2	8	0	13	209	400	458	513	485
Indiana	1	0	0	0	0	1	2	198	346	278
Iowa	5	0	0	0	0	5	7	6	6	76
Kansas	57	1	2	0	1	61	147	61	74	93
Kentucky	1	0	1	0	1	3	13	6	11	6
Louisiana	1	0	1	1	0	3	4	12	7	12
Maine	36	0	2	1	0	39	46	59	75	67
Maryland	7	0	0	0	0	7	211	169	110	123
Massachusetts	186	1	9	4	11	211	314	332	467	356
Michigan	31	0	6	1	4	42	83	126	107	100
Minnesota	36	0	3	0	3	42	165	152	143	89
Mississippi	0	0	0	0	0	0	2	0	1	0
Missouri	80	0	4	0	2	86	153	357	550	510
Montana	14	0	0	0	1	15	23	38	30	50
Nebraska	2	2	0	0	1	5	30	17	14	49
Nevada	0	0	0	0	0	0	1	0	1	0
New Hampshire	23	0	2	1	2	28	36	25	32	40
New Jersey	31	0	3	1	1	36	158	112	88	72
New Mexico	4	0	0	0	0	4	3	5	5	1
New York	99	0	2	0	4	105	1,114	1,491	1,630	1,371
North Carolina	3	1	0	1	0	5	8	8	3	4
North Dakota	3	0	2	0	0	5	5	2	8	11
Ohio	260	0	13	0	3	276	461	337	585	333
Oklahoma	8	0	2	0	0	10	49	32	43	101
Oregon	16	0	3	0	1	20	47	27	29	45
Pennsylvania	69	0	4	0	3	67	158	85	1,767	961
Rhode Island	122	0	5	1	3	141	229	181	210	381
South Carolina	4	0	0	0	0	4	0	2	3	0
South Dakota	14	1	1	0	1	17	21	14	8	9
Tennessee	0	0	0	0	0	0	6	6	9	7
Texas	40	2	2	0	1	44	42	21	60	22
Utah	5	0	1	0	0	6	1	5	18	31
Vermont	3	0	1	0	0	4	6	1	2	20
Virginia	16	1	0	0	1	18	11	6	13	20
Washington	27	0	4	1	6	38	32	66	70	134
West Virginia	3	0	0	1	0	4	4	0	41	28
Wisconsin	29	0	4	1	1	35	57	66	117	92
Wyoming	5	0	1	0	0	6	6	2	6	10
Unknown	9	0	1	0	0	10	10	0	0	37
	1,818	9	104	17	91	2,039	4,402	5,560	7,933	7,205

TETANUS

There were 2 cases of tetanus in both 1937 and 1938. In 1939 none were reported and in 1940 there were 2. In 1941 1 case of tetanus was reported from Massachusetts. In this case clinical symptoms of tetanus began eight days after a firecracker wound on the leg. Despite antitoxin administration as soon as diagnosis was made, the patient died.

SERIOUS INJURIES

Hospital questionnaires and newspaper clippings again record numerous serious and unusual injuries resulting from firecrackers. Nine persons lost the vision in one or both eyes and 104 sustained eye injuries. Nevertheless, as shown in table 2, there has been a decline in the total number of serious injuries.

INJURIES

Table 3 totalizes the injuries from firecrackers and other explosives in 1941 and in parallel columns the injuries recorded for the preceding four years. As has been pointed out in previous reports, these figures do not take into account hospitals which failed to report their accidents or injuries treated in physicians' offices. The totals, therefore, must be considered conservative and if all injuries were recorded the numbers would doubtless be considerably higher. The most striking figures in the report are those relating to New York State, where the injuries declined from over 1,100 in 1940 to 105 in 1941. This change unquestionably can be attributed to the state legislation, which first became effective on the Fourth of July of 1941. This year Ohio has the unenviable distinction of leading all states in the number of injuries, in spite of the fact that the total represents some decrease from previous years. Ohio is followed closely by California, Massachusetts and Illinois, in that order, all with bad records. Ohio passed an antifireworks bill to take effect August 1, 1941, which therefore was without value for this year. California has a limited law which was obviously not satisfactorily effective. In Massachusetts the antifireworks legislation was defeated. The large number of injuries in that state therefore can be directly attributed to failure to adopt legislation which was obviously indicated by experience of previous years. A bill to control fireworks will become effective in Illinois on Jan. 1, 1942.

However, the state with the worst record as judged by the relation between the number of injuries and the size of the population is Rhode Island. The states with the largest percentage of increase in injuries from 1940 to 1941 are Connecticut from 48 to 81, South

TABLE 4.—*Injuries in Principal Cities*

	1941*		1940		1939		1938		1937	
	Injuries	Rate per 100,000	Injuries	Rate per 100,000	Injuries	Rate per 100,000	Injuries	Rate per 100,000	Injuries	Rate per 100,000
Pawtucket, R. I.	25	33.3	...	....	....	....	....	....	....	....
Providence, R. I.	78	31.0	...	....	....	....	....	....	....	....
Youngstown, Ohio	48	28.7	...	....	....	....	....	....	....	....
Hartford, Conn.	46	27.7	...	....	....	....	....	....	....	....
Washington, D. C.	70	10.6	68	14.0	57	11.7	....	....	....	....
Boston	74	9.6	76	9.7	71	9.0	...	....	....	....
Los Angeles	132	8.8	124	10.0	258	20.9	180	14.6	45	3.6
Kansas City, Mo.	30	7.5	55	13.8	243	60.8	...	....	....	....
Cincinnati	26	5.7	62	13.7	31	6.9	...	....	....	....
Cleveland	47	5.4	64	7.1	42	4.7	129	14.3	64	7.1
Chicago	146	4.3	253	7.5	236	6.7	176	5.2	235	6.7
St. Louis	35	4.3	...	....	...	....	265	33.9	322	39.2

* Rate per hundred thousand based on 1940 census; rates for previous years based on 1930 census.

TABLE 5.—*Cap Pistol Injuries*

State	1941		
	Burns and Lacerations	Serious Injuries	Total
California	1	0	1
Maine	1	0	1
Massachusetts	1	0	1
Minnesota	2	1	3
Missouri	0	1	1
Pennsylvania	3	1	4
	8	3	11

Carolina from 0 to 4, Utah from 1 to 6 and Virginia from 11 to 18. Of these, apparently Utah is the only state with a satisfactory law on the books. Delaware is the only state which has had a perfect record of no injuries for the last two years. However, in 1941 Mississippi and Nevada did not record any fireworks accidents. Curiously, Tennessee recorded two deaths but no other fireworks injuries.

A striking improvement has occurred between 1940 and 1941 in Alabama, Arizona, Kansas, Kentucky, Maine, Nebraska, New Jersey and Oklahoma. Alabama, of course, as a Southern state, would be expected to have few fireworks accidents on the Fourth. The improvement in Arizona is probably due to the adoption of a control bill effective in 1941. The explanation for Kansas is unknown; Kentucky has a regulatory law. The excellent results obtained in Maryland are doubtless due to the adoption of a new law this year. No ready explanation for the improvement in Maine is available. Nebraska has had a law since 1937. New Jersey was one of the first states to adopt adequate legislation but showed from 1937 to 1940 a steady although slight rise in number of accidents, presumably due to laxity in enforcement. This year the trend has been strikingly reversed, and New Jersey shows the lowest number of injuries ever recorded for that state, this result being attributed in press releases to an embargo placed on all shipments of fireworks into the state and to the effectiveness with which the new law in New York was enforced. It is particularly noteworthy that Indiana, which had one of the worst records of any state up until the application of its antifireworks law in 1940, has for the last two years reported only three such accidents. Obviously, the states which need adequate state laws and enforcement most are Ohio, Massachusetts, Illinois and California. Of these Ohio and Illinois have laws which are to become effective for the Fourth of July 1942; it will be interesting to see how effective their enforcement will be. The record of Massachusetts with defeat of a satisfactory bill would imply an extraordinary lack of interest in the lives and limbs of its citizens.

Washington, D. C., leads the large cities in 1941 in the proportion of injuries per hundred thousand of population, displacing Baltimore, which, as a result of finally enacted and enforced legislation, no longer appears on the list of large cities with bad records. In table 4 appears a tabulation of the cities with the highest rates of injuries. It should be noted that four cities of intermediate size, two of them in Rhode Island, lead all the larger cities in rate of injuries per hundred thousand of population. It is noteworthy also that more than half of all the injuries which were reported in Connecticut occurred in one of its cities, Hartford.

This year hospitals were specifically instructed to list injuries from cap pistols separately, and these are given in table 5. Although this question was asked in 1940, comparable 1940 figures are not given since, because of the different method of collection, they presumably included some injuries from blank cartridges, guns and firecrackers. It is curious to note that, in spite of the small number of injuries, three were serious, in one case resulting in hospitalization for twenty-three days and in another for forty-two days. On close examination the majority of the injuries appeared to be due to the caps themselves rather than the normal use of the cap pistol.

COMMENT

As has been pointed out repeatedly in these reports, the reduction of fireworks injuries is dependent not only on adequate state legislation but on enforcement as well. In 1940 the state which demonstrated these points most dramatically was Indiana; this year Maryland and New York showed an enormous reduction in injuries. With the exception of most of the Southern states, which do not celebrate the Fourth of July with fireworks as pointed out recently in a letter from Mr. Bugbee, director of the National Fire Protection Association published in THE JOURNAL, there are few exceptions to the rule that only those states which have enacted and enforced statewide laws have shown evidence of satisfactory control. It is however a source of satisfaction that, in the five years since these reviews have been resumed, the total number of unnecessary accidents of this nature have declined from 7,205 in 1937 to 2,039 in 1941. This source of death and disfigurement can be still further reduced, especially by prompt enactment and enforcement of suitable legislation in those states, such as Massachusetts and Rhode Island, which do not have effective laws. Ohio and Illinois, if adequate enforcement is attained next year, should show considerable improvement. In view of the experience of the last two years the California legislation should receive prompt attention with a view to making it serve the purposes for which it was presumably intended.

Council on Physical Therapy

THE COUNCIL ON PHYSICAL THERAPY HAS AUTHORIZED PUBLICATION OF THE FOLLOWING REPORTS. HOWARD A. CARTER, Secretary.

ALLERGY ELECTRIC MASK NOT ACCEPTABLE

Manufacturer: Allergy Research Institute, Inc., P. O. Box 1399, Cincinnati.

The Allergy Electric Mask, a beak-shaped mask covering the nose and mouth, is claimed in the firm's advertising to be "An Advanced Scientific Treatment for Hay Fever—Rose Fever —Seasonal Asthma." It is said that the device ". . . will filter and purify the air inhaled by the wearer . . . 99.46% OF THE POLLENS AND MOLDS ARE REMOVED FROM THE AIR WITH THE ALLERGY ELECTRIC MASK." The perforated metal front rests on a rubber base which is fashioned to fit against the contour of the face; head straps hold the device in place.

In operation a mechanical method and an alleged electrical method of filtration are employed. The electric filter consists of two molded fine mesh metal screens, one attached to the negative and the other to the positive pole of a 1½ volt battery. The mechanical filter pad, a cellulose-sheet type filter, is placed between the two metal screens. Following is an excerpt from the firm's advertising:

> The ALLERGY ELECTRIC MASK causes the air to flow through a circuit containing a positive-charged screen and a negative-charged screen. Those pollens having a positive electrical charge are repelled by the positive-charged screen, while pollens having a negative electrical charge are repelled by the negative-charged screen. (A pollen attracted to an opposite-charged screen immediately loses its own charge and assumes the same charge as that of the screen, and is then repelled.)
>
> The Mask utilizes the electrical method of filtration to greatest advantage, because it contains a small battery which produces a complete electrical circuit about the filter pad. The positive pole of the battery, connected to a metal screen, gives this screen a positive charge. The negative pole of the battery, connected to the other screen, gives this other screen a negative charge. The filter pad lies between the two screens. As the air flows through the Mask, the harmful particles are removed by both mechanical and electrical methods of filtration.

The Council's investigation of the device brought forth the following results:

A series of exposures was made while the mask was worn by a patient for periods of a few hours each over a period of fifteen days. A petrolatum covered glass cover slip was placed both on the outside and on the inside of the mask and the number of pollens on the same area (usually 1.8 sq. cm.) were compared. The following results were obtained:

	Exterior	Interior
With Battery		
8/27	3	0
8/29	4	0
8/30	1	0
8/31	0	0
3/31	0	1
9/2	1	0
9/4	16	48
9/5	17	54
9/5	6	7
9/6	3	7
Without Battery		
9/7	18	11
9/8	15	17
9/9	1	2
9/10	0	1

In another experiment pollen was dispersed into a room by means of a fan and the mask with a cover slip on the outside, and one on the inside was worn by the experimenter for a period of thirty to sixty minutes. Pollen counts were then made as before, with the following results:

	Exterior	Interior
With Battery		
Experiment 1	58	82
Without Battery		
Experiment 2	142	121
Experiment 3	76	92

It is apparent not only that the filtering efficiency of the mask is poor but that the battery adds no advantage.

The Council voted not to include the Allergy Mask on its list of accepted devices because the claims made by the firm for the instrument were not substantiated by the Council's tests.

HAYRIN NASAL FILTER NOT ACCEPTABLE

Manufacturer: Allergy Research Institute, Inc., P. O. Box 1399, Cincinnati.

The Hayrin Nasal Filter is advertised as "A scientifically Designed and Specially Constructed (Invisible) Nasal Filter Which Will Aid in the Relief of Hay Fever, Rose Fever and Seasonal Asthma. . . ." It is manufactured and distributed by the firm manufacturing the Allergy Electric Mask and promotional matter from the firm states "The Allergy Electric Mask and the completely invisible Hayrin Nasal Filter make an ideal combination. The mask can be worn when sleeping, while driving, and when about the home; and the Filter can be worn at work, play and at social engagements."

Although the device was not submitted to the Council for consideration by the firm, the many inquiries coming to the office of the Council concerning the device made advisable the preparation of a report. The nasal filter was purchased and examined by the Council.

In the firm's promotional literature the device is claimed to "give an effect similar to carrying an air conditioning unit with you." The outfit consists of two silver frames into which are to be placed the filter pads. The frames are adjustable to varying sizes of nasal chambers. These filters are to be placed in the nose to filter out pollen, molds and other dust particles.

Many misleading statements and unjustifiable claims are made in the advertising and in the correspondence to a prospective purchaser. The filtering device is uncomfortable when fitted in the nose. Dry pollen placed on the filter pad did not go through the mesh. However, when such a pad is moistened with secretions it acts not only as an obstacle to pollen but also as an obstruction to the free entrance of air. Although the filter may stop pollen from entering the nose, it cannot prevent the entrance of pollen through the mouth and eyes. While such filters have in some instances improved the nasal symptoms they have not prevented, indeed have even favored, the occurrence of asthma.

The Council voted not to include the Hayrin Nasal Filter on its list of accepted devices.

Council on Pharmacy and Chemistry

NEW AND NONOFFICIAL REMEDIES

THE FOLLOWING ADDITIONAL ARTICLES HAVE BEEN ACCEPTED AS CONFORMING TO THE RULES OF THE COUNCIL ON PHARMACY AND CHEMISTRY OF THE AMERICAN MEDICAL ASSOCIATION FOR ADMISSION TO NEW AND NONOFFICIAL REMEDIES. A COPY OF THE RULES ON WHICH THE COUNCIL BASES ITS ACTION WILL BE SENT ON APPLICATION.

THEODORE G. KLUMPP, M.D., Secretary.

LIVER PURIFIED SOLUTION-DRUG PRODUCTS CO., INC.—A sterile aqueous, clear, dark brown solution containing all of the fraction G of the liver extract (Cohn), preserved with 0.5 per cent of carbolic acid (phenol). The daily parenteral administration of 0.1 cc. has been found to produce the standard reticulocyte response defined as 1 U. S. P. unit (injectable) when assayed in cases of pernicious anemia as required by the Council.

Actions and Uses.—Liver purified solution-Drug Products Co., Inc., is recommended for intramuscular use in the treatment of pernicious anemia. See the general article Liver and Stomach Preparations (New and Nonofficial Remedies, 1941, p. 328).

Dosage.—For the average case in relapse, 10 U. S. P. injectable units (1 cc.) may be injected as the initial dose. Subsequent doses from 5 to 10 U. S. P. injectable units (0.5 to 1.0 cc.) may be given weekly until the blood picture has returned to normal. The maintenance dose should be not less than 1 U. S. P. injectable unit (0.1 cc.) daily and should be adjusted to the needs of the individual patient depending on the degree of complications. It may be administered at weekly or longer intervals in equivalent cumulative doses.

Distributed by Drug Products Co., Inc., Long Island City, N. Y.
Hyposols Liver Purified Solution-Drug Products Co., Inc., 1 cc. (10 U. S. P. injectable units per cubic centimeter).
Vials Liver Purified Solution-Drug Products Co., Inc., 10 cc. (10 U. S. P. injectable units per cubic centimeter.)

Liver purified solution-Drug Products Co., Inc., 10 units per cubic centimeter, is prepared as follows: Fresh edible liver is extracted with water at 170 F. for thirty minutes and filtered. The filtrate is concentrated in vacuo and extracted with 70 per cent alcohol; the alcoholic extracts are concentrated in vacuo and precipitated with ammonium sulfate. The precipitate is further purified by alcoholic fractionation, the alcohol removed and the extract made up to volume so that each cubic centimeter contains the extract from 100 Gm. of fresh liver. Five-tenths per cent phenol is used as a preservative.

SOLUTION OF POSTERIOR PITUITARY (See New and Nonofficial Remedies, 1941, p. 395).

PITUITARY SOLUTION-U. S. P. (LAKESIDE).—A brand of solution of posterior pituitary-U. S. P.

Manufactured by Lakeside Laboratories, Inc., Milwaukee.
Ampul Pituitary Solution-U. S. P., 0.5 cc. (Lakeside).
Ampul Pituitary Solution-U. S. P., 1 cc. (Lakeside).
Vials Pituitary Solution-U. S. P., 10 cc. (Lakeside).
Vials Pituitary Solution-U. S. P., 30 cc. (Lakeside).

ALUMINUM HYDROXIDE GEL (See THE JOURNAL, Nov. 1, 1941, p. 1539).

Aluminum Hydroxide Gel-Schieffelin.—A brand of aluminum hydroxide gel-N. N. R. The preparation contains 5.5 per cent aluminum hydroxide (equivalent to 3.3 per cent aluminum oxide). Saccharin and Oil of Peppermint U. S. P. are added as flavoring agents. Marketed in bottles of 1 pint and 1 gallon.

Manufactured by Schieffelin & Co., New York. No U. S. patent or trademark.

MERCURIC SALICYLATE (See New and Nonofficial Remedies, 1941, p. 349).

Cheplin Biological Laboratories, Inc., Syracuse, N. Y.

Bottles Mercuric Salicylate, 1 grain (0.065 Gm.) Suspended in Oil, 60 cc.: Each cubic centimeter contains mercuric salicylate 1 grain (0.065 Gm.), quinine and urea hydrochloride 0.005 Gm., wool fat 0.1 Gm., double distilled water 0.05 cc. and Wesson oil (corn oil) to make 1 cc.

NICOTINIC ACID (See New and Nonofficial Remedies, 1941, p. 555).

The following dosage forms have been accepted:

Tablets Nicotinic Acid, 20 mg., 100 mg.
Prepared by George A. Breon & Company, Inc., Kansas City, Mo.

SULFANILAMIDE (See New and Nonofficial Remedies, 1941, p. 506).

The following dosage forms have been accepted:

Tablets Sulfanilamide, 2½ grains, 5 grains, 7½ grains.
Prepared by McNeil Laboratories, Inc., Philadelphia.

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SATURDAY, JANUARY 3, 1942

A CALL TO THE MEDICAL PROFESSION

Last week The Journal published an enrolment form for the Procurement and Assignment Service for Physicians. Already many physicians have sent this blank directly to the headquarters of the Procurement and Assignment Service in Washington. On the opposite page appears another copy of the enrolment form. EVERY PHYSICIAN IN THE UNITED STATES WHO HAS NOT YET SENT THIS FORM TO THE EXECUTIVE OFFICER IN WASHINGTON SHOULD DO SO AT ONCE.

The only way in which the Procurement and Assignment Service can function to supply the Army, Navy and Public Health Agencies with the necessary personnel and to make sure that civilian and industrial needs are suitably cared for is to have the complete cooperation of the medical profession. FILL OUT THE BLANK ON THE OPPOSITE PAGE, TEAR IT OUT OF THE JOURNAL AND SEND IT AT ONCE TO DR. SAM F. SEELEY, EXECUTIVE OFFICER OF THE PROCUREMENT AND ASSIGNMENT SERVICE.

If you have already sent in the blank published last week, make certain that some other physician who may not have received a blank does the same. Ask your professional colleagues whether or not they have enrolled. If they have not yet enrolled, tell them to send in the blank which is made available through The Journal of the American Medical Association or through a state medical journal and offer them the extra blank published in this week's issue if they do not have one available.

MECHANISM OF ANTIPNEUMOCOCCUS SERUM THERAPY

Studies of the cytologic mechanism of spontaneous recovery from experimental pneumococcic pneumonia in rats and modification of this mechanism under the influence of type specific antiserum are currently reported by Wood [1] of the department of bacteriology and immunology at the Harvard Medical School. Since experimental pneumococcic pneumonia in rats closely simulates human lobar pneumonia, the results of the investigation are presumably applicable to clinical medicine. Previous studies of clinical and experimental pneumococcic pneumonia have led to numerous contradictions and inconsistencies. Fully encapsulated living pneumococci are recognized to be resistant to attack by phagocytic cells unless previously sensitized with specific antibodies.[2] Nevertheless, investigators have shown that complete recovery may occur both in man and in laboratory animals long before specific antibodies can be demonstrated in the circulation.[3] Well supported laboratory and clinical evidence indicates that intravenous injection of specific antiserum often brings about a rapid recovery, yet much evidence has been advanced that specific antibodies cannot penetrate the pneumonic lung.[4] To complicate matters, quantitative data indicate that, were antibodies able to enter the pulmonary alveoli, they could not accumulate in sufficient amounts to neutralize the antiphagocytic type specific polysaccharide there present. Nye and Harris,[5] for example, estimate that at least 60 liters of the highly potent type I antiserum would be necessary to precipitate completely the inhibiting pneumococcus carbohydrate. Drugs of the sulfapyridine group are not bactericidal in concentrations usually employed in clinical medicine; nevertheless, following treatment with sulfapyridine, patients often recover without the aid of circulatory antibodies.[6]

In order to settle these and other controversial questions, Wood made a detailed microscopic study of the evolution of experimental pneumococcic pneumonia in rats. By means of an adaptation of the technic of Jourdonais and Nungester [7] the rats were inoculated by intrabronchial injection of 0.1 cc. of 6 per cent mucin containing from 4,000 to 7,000 highly virulent type I pneumococci. To insure alveolar penetration of the inoculum, the rats were hung in a vertical position by the upper incisor teeth for thirty minutes while still under light ether anesthesia.

The pneumococcic pneumonia produced by this technic is uniformly fatal. All 40 untreated control rats

1. Wood, W. B., Jr.: J. Exper. Med. **73**: 201 (Feb.) 1941.
2. Robertson, O. H., and Van Sant, Helen: J. Immunol. **37**: 571 (Dec.) 1939.
3. Lord, F. T., and Persons, E. L.: J. Exper. Med. **53**: 151 (Feb.) 1931.
4. Kline, B. S., and Winternitz, M. C.: J. Exper. Med. **21**: 311, 1915. Fox, J. P.: J. Immunol. **31**: 7 (July) 1936.
5. Nye, R. N., and Harris, A. H.: Am. J. Path. **13**: 749 (Sept.) 1937.
6. Wood, W. B., Jr., and Long, P. H.: Ann. Int. Med. **13**: 612 (Oct.) 1939.
7. Jourdonais, L. F., and Nungester, W. J.: Science **81**: 74 (Jan. 18) 1935.

ENROLMENT FORM FOR PROCUREMENT AND ASSIGNMENT SERVICE FOR PHYSICIANS

Dr. Sam F. Seeley, Executive Officer
Procurement and Assignment Service
New Social Security Building
4th and C Streets S.W.
Washington, D. C.

Dear Doctor Seeley:

Please enroll my name as a physician ready to give service in the Army or Navy of the United States when needed in the current emergency. I will apply to the Corps Area commander in my area when notified by your office of the desirability of such application.

Signed _____

1. Give your name in full, including your full middle name:

2. The date of your birth:

3. The place of your birth:

4. Are you married or single?

5. Have you any children? If so, how many?

6. Do you believe yourself to be physically fit and able to meet the physical standards for the Army and Navy Medical Corps?

7. Have you filled out previously the questionnaire sent to all physicians by the American Medical Association?

8. When and where were you graduated in medicine?

9. In what state are you licensed to practice?

10. Do you now hold any position which might be considered essential to the maintenance of the civilian medical needs of your community? If so, state these appointments:

11. Have you previously applied for entry into the Army or Navy Medical Service? If so, state when, where and with what result (if rejected, state why).

Signature _____

Date _____ Address _____

Fill out this blank, tear out and send to Dr. Sam F. Seeley at the above address.

succumbed to the disease in less than five days, the majority dying in about forty-eight hours. Necropsies showed consolidation of the lobar type, usually with an accompanying pleurisy, pericarditis and bacteremia.

To study the evolution of the pulmonary lesion, groups of 5 untreated rats were killed at the end of twenty minutes, two hours, six hours, twelve hours, eighteen hours, twenty-four hours and thirty-six hours after inoculation. Their inflated lungs were fixed in situ. A composite picture of all groups showed a small circumscribed area of consolidation in the injected pulmonary lobe at the end of two hours, increasing to an involvement of the entire lobe by the end of thirty-six hours. Three definite histologic zones could be recognized in this spreading lesion: First there was an advancing outer zone characterized by the presence of edema fluid in the alveoli. This edema fluid contained few if any leukocytes but did contain innumerable pneumococci, which apparently multiplied freely in this favorable medium. Outward mechanical flow of this infected edema fluid was apparently the main mechanism of spread of the pulmonary lesion.

Inside this peripheral zone there was a second zone in which the alveoli contained both leukocytes and micro-organisms. In the outer portion of this intermediate zone, where the leukocytes were relatively few, pneumococci were numerous. In the inner portion the leukocytes dominated and bacteria were scarce. Phagocytosis of the pneumococci by polymorphonuclear leukocytes was a conspicuous feature of this portion, even though specific opsonins were not demonstrable at this stage. Finally there was an inner zone of advanced consolidation characterized by the complete absence of organisms, the alveoli being packed with leukocytes and fibrin deposits being occasionally observed. In the older parts of the consolidated area, macrophagic resolution had already begun.

The histopathology of the forty-eight hour lungs was essentially the same, except that by this time the lesion had usually extended to adjacent lobes. In all stages the essential site of the spreading infection was in the outer edema zone, with complete or practically complete leukocytic sterilization in the consolidated areas. To explain this phagocytosis it must be assumed either that specific opsonins are formed locally in the infected tissues [8] or that there are unknown local hormonal or enzymic factors making possible adequate phagocytosis in the absence of specific opsonins.

Turning now to a study of the therapeutic effects of specific antiserum, Wood treated groups of infected rats with homologous type-specific antipneumococcus serum. Deaths did not occur among the 76 rats treated within eighteen hours after intrabronchial inoculation. Conditions in animals killed from four to seven days later indicated in each case that the pneumococcic infection had completely subsided. To determine the mechanism of this therapeutic recovery, groups of treated animals were killed at various times after treatment. Within six hours after peripheral injection of the antiserum the spreading lesion was arrested, the outward margin becoming sharply demarcated. Within one hour after injection of the antiserum the pneumococci of the edema zone could be seen to be agglutinated and firmly adherent to the alveolar walls. By the end of six hours, practically all of the extracellular organisms of this zone had congregated into large immobilized clumps. This included pneumococci in large bronchi as well as within the alveoli. The clumped cocci often showed capsular swelling. Accelerated phagocytosis was noted in all cases. The essential mechanism of the therapeutic arrest is therefore apparently agglutination and immobilization of the pneumococci by alveolar adhesion, presumably due to specific epithelial opsonins analogous to the "endothelial opsonins" of earlier investigators.[9] This peripheral immobilization apparently allows the micro-organism to be overtaken and destroyed by the advancing leukocytes. Within forty-two hours practically all pneumococci had been engulfed and completely digested by the phagocytic cells.

In contrast to this favorable action, Wood found that, if the administration of specific antiserum was delayed more than twenty-four hours after intrabronchial inoculation, nearly half of the treated animals died within eighteen hours, some of them even during the course of the serum injection. A definite explanation of this therapeutic shock is not suggested, though the animals evidently died of an anaphylactic-like reaction. Each rat showed evidence of pulmonary edema at the time of death, frothy hemorrhagic fluid exuding from the nostrils.

It has been suggested [10] that, owing to its relatively small molecular diameter, rabbit antibody would presumably penetrate lung tissues more readily than horse serum. Wood found antipneumococcus horse serum equally effective with rabbit antiserum. Microscopic examination of type I pneumococcic lungs treated with type II antiserums showed that heterologous antibody is without demonstrable therapeutic effect. Preliminary experiments have already shown that sulfapyridine causes a similar peripheral immobilization of pneumococci, though the mechanism of this immobilization is apparently different from that with specific antiserum. Further studies of this chemotherapeutic peripheral immobilization are now in progress in the Harvard laboratories.

8. Robertson, O. H.: Recent Studies on Experimental Lobar Pneumonia: Pathogenesis, Recovery and Immunity, J. A. M. A. **111**: 1932 (Oct. 15) 1938.

9. Manwaring, W. H., and Coe, H. C.: J. Immunol. **1**: 401, 1916.

10. Horsfall, F. L., Jr.; Goodner, Kenneth; MacLeod, C. M., and Harris, A. H., 2d: Antipneumococcus Rabbit Serum as Therapeutic Agent in Lobar Pneumonia, J. A. M. A. **108**: 1483 (May 1) 1937.

Current Comment

OPPORTUNITIES FOR PHYSICIANS

Much has been said about the rapid advance of medical science and the relatively slower march of medical practice—the lag between the acquisition of knowledge and its utilization. In order to shorten the time required for dissemination throughout the profession of information concerning the practical application of the newest discoveries and the latest improvements in the technic of diagnosis and treatment, a network of continuation courses for practicing physicians has been established under the auspices of medical societies, health departments, medical schools, hospitals and other agencies. Varying widely in methods employed, time involved and geographic distribution, these courses are designed to increase the knowledge and skill of those responsible for the care of the sick. For those who would avail themselves of these opportunities, the Council on Medical Education and Hospitals has compiled and publishes in this issue of THE JOURNAL a classified list of courses of five days' duration or longer which will be offered during the next three months. The number and the scope of these offerings present substantial evidence that the medical profession is alive to its responsibilities.

DETECTION OF EARLY OCULAR CHANGES IN AVITAMINOSIS A

Assessment of the nutritional status of human beings has long been a subject of study; particularly, in recent years, new methods and technics have been devised and older ones improved to provide more accurate means of evaluation. Early recognition of impairment due to dietary deficiencies is important. Kruse [1] recommends a biomicroscopic ocular examination as a simple and objective method for the detection of xerosis conjunctivae, an early sign of vitamin A deficiency. An application of this type of study to a hundred and fifty adults from a low income group showed that in 45 per cent of the subjects ocular manifestations in the form of characteristic elevated conjunctival spots, which are a feature of advanced xerosis conjunctivae, could be detected by gross examination. Another 54 per cent had microscopic ocular lesions characteristic of avitaminosis A. Vitamin A therapy effected complete disappearance of the conjunctival lesions in some of the patients; in all others who were still receiving vitamin A at the time of the report the lesions had receded to the point of near disappearance. Months of therapy, however, were required to effect complete recovery. As interpreted by Kruse, the results of this limited survey revealed an unusually high prevalence of avitaminosis A in a low income group. The question arises concerning the relative value of this examination as compared with others depending on a measurement of dark adaptation.[2] Fortunately, a study on the correlation of conjunctival lesions with dark adaptation has been carried out, and a report of this work is forthcoming. Pertinent is the suggestion of Kruse that xerosis probably precedes night blindness as an early sign of avitaminosis A. Further research will undoubtedly elucidate this important point and will contribute to the broader problem of the early detection of avitaminosis A.

1. Kruse, H. D.: Medical Evaluation of Nutritional Status: IV. The Ocular Manifestations of Avitaminosis A, with Especial Consideration of the Detection of Early Changes by Biomicroscopy, Pub. Health Rep. **56**: 1301 (June 27) 1941.

2. Dark Adaptation and Vitamin A, Current Comment, J. A. M. A. **117**: 370 (Aug. 2) 1941.

MERCURIALISM IN THE FELT HAT INDUSTRY

A solution containing mercury has long been used to treat fur in order to make it amenable to felting. In this "carroting" process part of the metal enters into chemical combination with the fur, from which it then gradually volatilizes into the air as elemental mercury. A serious health hazard is thus created. A recent survey by the U. S. Public Health Service revealed that over 10 per cent of a group of several hundred hatters examined were suffering from chronic mercurialism, a syndrome which is characterized by fine intention tremor, psychic irritability of an exaggerated degree, exaggerated tendon reflexes, pallor, and certain abnormalities of the mouth, among other symptoms.[1] A serious effort has been made to eliminate the use of mercury in the treatment of fur by the development of mercury free agents. Of interest in this connection is a reprt by Beal, McGregor and Harvey [2] in which is described the theoretical and the practical work culminating in the successful use of nonmercurial carroting materials. From the end of 1936 to September 1941 some 2,000,000 pounds of fur was carroted with these agents and in 1941 alone it may be estimated that 1,500,000 pounds was thus treated. Owing to the introduction of carrots free from mercury, it seems possible that mercurialism may in the future be entirely banished from the felt hat industry. This development has been greatly assisted by the industry itself. Investigations which have resulted in the development of nonmercurial carroting agents were promoted by the industry through the establishment of a fellowship at the Mellon Institute—an excellent illustration of how planned research may help to eliminate industrial health hazards. While we can look forward to the day when the felt hat industry is no longer plagued with occupational disease, the danger is present wherever mercury is handled. In any case, however, the mercury hazard can be minimized by following the suggestions of the bureau of public health, which recommends the segregation of sources of volatile mercury and ventilation so arranged as to minimize exposure of workers to mercury vapor.[1]

1. Neal, P. A.; Flinn, R. H.; Edwards, T. I.; Reinhart, W. H.; Hough, J. W.; Dalla Valle, J. M.; Goldman, F. H.; Armstrong, D. W.; Gray, A. S.; Coleman, A. L., and Postman, B. F.: Mercurialism and Its Control in the Felt Hat Industry, Pub. Health Bull. 263, U. S. P. H. S., Government Printing Office, Washington, D. C., 1941.

2. Beal, G. D.; McGregor, R. R., and Harvey, A. W.: Elimination of Mercury Hazard in the Felt Hat Industry, Indust. & Engin. Chem., News Ed. **19**: 1239 (Nov. 25) 1941.

MEDICAL PREPAREDNESS

In this section of The Journal each week will appear official notices by the Committee on Medical Preparedness of the American Medical Association, announcements by the Surgeon Generals of the Army, Navy and Public Health Service, and other governmental agencies dealing with medical preparedness, and such other information and announcements as will be useful to the medical profession.

CAMPAIGN FOR CORRECT DIET FOR CIVILIAN DEFENSE

Representatives of the federal government, the municipal health department, the organized medical, dental and nursing professions, food industries and welfare agencies addressed a meeting in the Health Building, New York City, December 10, which opened a campaign to encourage the use of correct diets as an essential phase of civilian defense. The health commissioner of New York City, Dr. John L. Rice, who presided, said that the principal objectives of the program were to see that no one lacks sufficient food; second, that the wastage of food in homes and restaurants be stopped, and, third, to prevent the hoarding of food. The acting mayor, Newbold Morris, pointed out that the problem was mainly one of education and recommended as educational material a booklet prepared for distribution by the city health department entitled "Food Joins the Colors." Dr. W. H. Sebrell, deputy assistant director of the Federal Security Agency in charge of nutrition, pledged the support of various coordinating bureaus of the federal government in the nutrition program in the various cities. Assistant Superintendent of Schools William Jansen said that plans had already been worked out for the mass feeding of children that might have to leave New York City. Deputy Welfare Commissioner Leo Arnstein said that his department aided the needy to get proper diet not only by direct cash assistance but also through three thousand investigators of the department giving clients on relief advice on nutrition. City Markets Commissioner William F. Morgan Jr. said that no bombing of the New York City area would cause serious food shortage, as the most exact information exists on what food and how much is handled at each market and depot, and preparations have been made to reroute food deliveries in case of bombings. Among the other speakers were Dr. Maximilian A. Ramirez, president-elect of the New York County Medical Society; Dr. Adam Eberle, general medical superintendent of the hospitals department; Dr. John O. McCall, director of the Guggenheim Dental Clinic, and Clarence Francis, president of the General Foods Corporation.

RECREATION CENTERS AND SERVICE CLUBS

The army has built one hundred and nine service clubs on military reservations throughout the country to provide the recreation and social advantages of a men's club for enlisted personnel; twenty-two others are under construction, while ninety-three guest houses are already operating and nineteen others have been authorized. These clubs are designed for recreational reading, refreshments, musicals, parties, dances and as a meeting place for enlisted men, their families and friends. Accommodations for overnight guests and quarters for hostesses are furnished in guest houses constructed nearby as an adjunct to the service clubs, which are of three general types, depending on the size of the garrison. The largest type club has three hostesses and one librarian. An allotment of $10,500 was made to equip each of the medium sized (SC-4) clubs. Guests occupying the guest houses are charged a nominal sum to defray the cost of laundering linens and necessary housekeeping service. The usual cost per person is 50 cents a night.

Paul V. McNutt, director of defense health and welfare services, announced on November 28 completion of construction at Fayetteville, N. C., of the first two of several hundred service men's recreation centers. The Fayetteville centers are among the two hundred and seventy-three constructions and renovations being provided with federal money through the Community Facilities Act, in communities near army camps and naval posts, almost all of which were to be finished by New Year's. Fayetteville, with a normal population of 18,000, is 10 miles from Fort Bragg, where the garrison amounts to nearly seventy thousand; during the maneuvers about one hundred and twenty thousand soldiers were in that area. The Fayetteville Defense Recreation Council, cooperating with the Federal Security Agency, has undertaken to coordinate community recreation, utilizing facilities in schools, churches and private homes, which, however, in themselves were insufficient.

UNNECESSARY AMBULANCE CALLS AND THE SHORTAGE OF INTERNS

The commissioner of hospitals of New York City, Dr. Willard C. Rappleye, announced on Dec. 21, 1941 that it probably would be necessary in the near future to discontinue interns on the ambulances of the voluntary and municipal hospitals, owing to the increasing shortage of interns. Because of the need of medical officers for the expanding army and navy, many interns will be called to military duty.

Under the plan contemplated, trained attendants competent to deal with first aid and emergency care will be provided on each ambulance. All patients requiring diagnosis or medical attention will be promptly brought to the hospital, where they will be seen in the emergency ward and proper diagnosis and treatment established. Those who require hospitalization will be admitted; those who require only temporary care will be kept in the emergency ward until they can be discharged.

Dr. Rappleye stated that the Department of Hospitals and the voluntary institutions have for months been working on plans to provide trained ambulance attendants to take the place of interns on ambulances. In most large cities the ambulance service is manned by nonmedical personnel. At present in New York City less than half of the ambulance calls are for emergencies; many of the calls are for medical attention in the home or for minor injuries which could be taken care of in an outpatient department or by a neighborhood physician. Commissioner Rappleye appeals to individuals, families and community organizations to cooperate by going to private physicians or to outpatient departments and hospitals for medical attention rather than calling ambulances to their homes for ordinary illnesses or minor complaints.

The program now being studied would not change the medical and nursing personnel in the eighty emergency units now organized throughout the city that are standing by for any major catastrophe in the community.

LABORATORY AIDS IN DIAGNOSIS OF NEUROTROPIC VIRUS DISEASES

An item was printed in this section of THE JOURNAL, November 8, page 1631, concerning laboratory aids in the diagnosis of neurotropic virus diseases. In this circular letter from the Office of the Surgeon General, U. S. Army, it was stated that the Virus Laboratory of the Army Medical School would undertake to perform special diagnostic tests on specimens submitted from the field. It is apparent now that civilian physicians and hospitals gained the impression that this army laboratory service would be available for their use, as numerous requests from these sources for further information about this service has been received at the Army Medical School. The Surgeon General's Office desires to announce that this special laboratory service cannot be made available to civilians at this time.

ARMY RESERVE OFFICERS ORDERED TO ACTIVE DUTY

SIXTH CORPS AREA

The following additional medical reserve corps officers have been ordered to extended active duty by the Commanding General, Sixth Corps Area, which comprises the states of Wisconsin, Illinois and Michigan:

ALPINER, Sam, 1st Lieut., Battle Creek, Mich., 1609th Corps Area Service Unit, Fort Custer, Mich.
DOLAN, Patrick T., 1st Lieut., Pittsfield, Ill., 5th Division, Fort Custer, Mich.
GOODMAN, Daniel H., 1st Lieut., Galesburg, Ill., Reception Center, Camp Grant, Ill.
HERBERT, Walter N., 1st Lieut., Yale, Mich., Reception Center, Fort Custer, Mich.
KANTER, Lester, 1st Lieut., Chicago, 5th Infantry Division, Fort Custer, Mich.
KETTERER, Francis H., 1st Lieut., Breese, Ill., Reception Center, Scott Field, Ill.
KOVEN, Arthur Jerome, 1st Lieut., Maywood, Ill., Reception Center, Fort Custer, Mich.
LEAVITT, Arnold H., 1st Lieut., Chicago, Reception Center, Fort Custer, Mich.
MUNCH, Robert F., 1st. Lieut., Chicago, Reception Center, Camp Grant, Ill.
POMARANC, Mark M., 1st Lieut., Chicago, Station Hospital, Camp McCoy, Wis.
PUTRA, Anthony Marion, 1st Lieut., Detroit, 33d Division, Camp Forrest, Tenn.
REID, Stephen E., 1st Lieut., Chicago, Station Hospital, Camp Grant, Ill.
ROBBINS, Fred Phillip, 1st Lieut., Chicago, Will Rogers Field, Oklahoma City.
WEISBERG, William Wiener, 1st Lieut., Chicago, Reception Center, Scott Field, Ill.
WINSAUER, Henry John, 1st Lieut., Kohler, Wis., Fred Harman, Ballinger, Texas.
YESINICK, Louis, 1st Lieut., Chicago, 1609th Corps Area Service Unit, Fort Custer, Mich.
ZIMMERMAN, Nathan, 1st Lieut., Chicago, Parks Air College, East St. Louis, Ill.

Orders Revoked

ESPENSCHEID, John S., 1st Lieut., Danville, Ill., Parks Air College, East St. Louis, Ill.
LEPPERT, Charles L., 1st Lieut., Rockford, Ill., Reception Center, Camp Grant, Ill.

SEVENTH CORPS AREA

The following additional medical reserve corps officers have been ordered to extended active duty by the Commanding General, Seventh Corps Area, which comprises the states of North Dakota, South Dakota, Minnesota, Nebraska, Iowa, Kansas, Missouri, Arkansas and Wyoming:

HARVEY, George, Jr., 1st Lieut., Rochester, Minn., Corps Area Service Command Station Hospital, Fort Snelling, Minn.
JERNIGAN, James Powell, 1st Lieut., Little Rock, Ark., Corps Area Service Command Station Hospital, Camp J. T. Robinson, Ark.
MILLER, Richard White, 1st Lieut., Fayetteville, Ark., Corps Area Service Command Station Hospital, Camp J. T. Robinson, Ark.
OTTEN, Donald Earnest, 1st Lieut., Minneapolis, Corps Area Service Command Induction Station, Fort Snelling, Minn.
REDMOND, William Storey, 1st Lieut., St. Joseph, Mo., Corps Area Service Command Station Hospital, Fort Leonard Wood, Mo.
STUMP, Robert Byron, 1st Lieut., Iowa City, Corps Area Service Command Station Hospital, Fort Leonard Wood, Mo.
SWANSON, Vincent Francis, 1st Lieut., Rochester, Minn., Corps Area Service Command Station Hospital, Fort Snelling, Minn.

Orders Revoked

STAUFFER, Harry B., Major, Jefferson City, Mo., Station Hospital, Camp J. T. Robinson, Ark.

Relieved from Active Duty

CHUNN, Stanley Sylvester, 1st Lieut., Pipestone, Minn., Fort Omaha, Neb.
SMAZAL, Stanley Francis, 1st Lieut., Davenport, Iowa, Fort Des Moines, Iowa.

NINTH CORPS AREA

The following additional medical reserve corps officers have been ordered to extended active duty by the Commanding General, Ninth Corps Area, which comprises the states of Washington, Montana, Oregon, Nevada, Utah, California and Idaho:

ANDERSON, Edward R., 1st Lieut., Tacoma, Wash., Fort Lewis, Wash.
BELL, Lloyd T., 1st Lieut., Culver City, Calif., Camp Callan, Calif.
BROOKE, Joseph M., 1st Lieut., Ronan, Mont., Station Hospital, Fort Lewis, Wash.
CHONG, Harry S., 1st Lieut., Sacramento, Calif., West Coast Air Corps Training Center, Moffett Field, Calif.
COHEN, Sidney, 1st Lieut., Provo, Utah, 7th Division, Fort Ord, Calif.
HAMILTON, Robert L., Captain, Marysville, Calif., Camp San Luis Obispo, Calif.
HARRIS, Chester S., Colonel, San Francisco, Recruiting and Induction Board, San Francisco.
JACOBSON, Hjalmer A., Captain, Fresno, Calif., Camp Roberts, Calif.
KAY, Raymond M., 1st Lieut., Van Nuys, Calif., Letterman General Hospital, San Francisco.
OWYANG, Edwin, 1st Lieut., San Francisco, Medical Replacement Training Center, Camp Grant, Ill.
SAYLIN, Joseph, Colonel, Pacific Palisades, Calif., Camp Roberts, Calif.
WENDLE, Cornelius C., 1st Lieut., Sand Point, Idaho, Station Hospital, Fort Lewis, Wash.

Relieved from Active Duty

CLOTHIER, William L., 1st Lieut., Camp Callan, Calif.
COLLINGS, Maurice M., 1st Lieut., Fresno Air Base, Calif.
FISHER, Louis J., 1st Lieut., 53d Evacuation Hospital, Camp San Luis Obispo, Calif.
GARDENIER, William H., 1st Lieut.
GOURSON, Nathaniel E., 1st Lieut.
KOSTICHEK, Robert J., 1st Lieut., 7th Division, Fort Ord, Calif.
McDONALD, John B., 1st Lieut., Camp San Luis Obispo, Calif.
MILLER, Ernest C., 1st Lieut., McChord Field, Wash.
SASLAW, Lewis B., Captain.

ORDERED TO FOREIGN DUTY

BARALD, Fred Charles, 1st Lieut., New Haven, Conn., Station Hospital, Losey Field, Ponce, Puerto Rico.
BELZ, Joseph Aloysius, Captain, 25th Division Artillery Dispensary, Schofield Barracks, Honolulu, Hawaii.
BRENNAN, John James, 1st Lieut., Post of Manila, Manila, Philippine Islands.
CHESLER, William, 1st Lieut., Philadelphia, 70th Medical Battalion, Fort Clayton, Canal Zone.
CORRADO, Albert Guy, 1st Lieut., Connellsville, Pa., Fort Amador, Balboa, Canal Zone.
COUNTER, Henry Milton, 1st Lieut., Ladd Field, Fairbanks, Alaska.
CULVER, Wesley Youngs, 1st Lieut., Westhampton Beach, N. Y., Fort Clayton, Canal Zone.
DAY, Romney Maxwell, 1st Lieut., Albrook Dispensary, Albrook Field, Canal Zone.
DETER, Dwight Meyer, Captain, Austin, Texas, Station Hospital, Fort Mills, Corregidor, Philippine Islands.
FLAIG, Julian Vincent, Captain, Station Hospital, Schofield Barracks, Honolulu, Hawaii.
GLUKFELD, Jerome Phillip, 1st Lieut., San Francisco, Fort Richardson, Alaska.
GOLD, Jacob Louis, Captain, Brooklyn, Schofield Barracks, Honolulu, Hawaii.
GOLDBERG, Joseph Dancis, 1st Lieut., New York, Station Hospital, Schofield Barracks, Honolulu, Hawaii.
GROSSMAN, Aaron, Captain, Department Training Center, Rio Hato, Albrook Field, Canal Zone.
HAYNES, Henry Matthais, Jr., 1st Lieut., Gatesville, Texas, Yakutat Landing Field, Yakutat, Alaska.
JONES, Leonard Bonham, 1st Lieut., Taft, Texas, Station Hospital, Fort Greely, Kodiak, Alaska.
KENDIG, Tom Atkins, 1st Lieut., San Antonio, Texas, Station Hospital, Fort Greely, Kodiak, Alaska.
KRAJESKI, Romuald John Frank, 1st Lieut., Wilkes-Barre, Pa., Panama Canal Department, Quarry Heights, Balboa Heights, Canal Zone.
MORTON, John Buck, 1st Lieut., Gray, Ga., Department Training Center, Rio Hato, Balboa Heights, Canal Zone.
MUSSELMAN, Merle McNeil, 1st Lieut., Omaha, Station Hospital, Fort Stotsenburg, Philippine Islands.
NICOSIA, Ralph Vincent, 1st Lieut., Houston, Texas, Tripler General Hospital, Honolulu, Hawaii.
TOWNSEND, Frank Marion, 1st Lieut., Harwood, Texas, Panama Coast Artillery Command, Fort Amador, Balboa, Canal Zone.
WANNER, Jay George, Captain, Medical Detachment, 2d Battalion, 37th Infantry, Fort Greely, Kodiak, Alaska.

ORGANIZATION SECTION

MEDICAL ECONOMIC ABSTRACTS

REAL LIFE TABLES EXCEED HYPOTHETICAL

In 1922 and again in 1933 the Metropolitan Life Insurance Company prepared hypothetical life tables to show where improvement in mortality might be expected. The census bureau has now issued a similar table based on the experience of white females in the general population of the United States for the period 1930 to 1939. The remarkable fact is that this table, based on actual experience, is almost identical with the hypothetical ones prepared almost twenty years earlier. "Except for the first year of life and at ages 50 and 60," says the Metropolitan Statistical Bulletin for November 1941, "the actual death rates for white females in 1930-1939 proved to be lower than in the hypothetical table of 1922. Thus have the hopes of the hypothetical table been not merely realized but surpassed by actual experience—and all this within two decades."

Another hypothetical table was prepared in 1933 based essentially on certain assumed reductions from the mortality of New Zealand females for 1931. Already actual experience is surpassing this new hypothetical table in some respects. "We have reason to believe," says the Bulletin, "that when all the data from our 1940 census are in it will be found that large sections of this country, principally in the Midwest, have mortalities as favorable as those of New Zealand." A new hypothetical table is again being prepared which starts with an expectation of life at birth of 70.78 years.

EXPLOITING MEDICAL SERVICE PLANS

The British Columbia Medical Association has for some time operated a prepayment plan much like the plans conducted by several of the state medical associations in the United States. Contracts have been made with industrial and other groups and the physicians have agreed to accept a reduction of 25 per cent from their regular fee schedules in payment for services rendered to those enrolled under the plan.

Within recent months, according to the Bulletin of the Vancouver Medical Association for November 1941, a number of quasi-insurance companies with various names have arisen. These undertake the sale of policies purporting to insure the beneficiary against the costs of sickness. When the patient has had an operation, the bill is sent to the patient for the full amount of the regular fee schedule. The organization then deducts 25 per cent of this bill and sends a check for the remainder to the physician. There is no contract between these companies and the medical association to accept any such reduction in fees. While the contracts with groups under the medical association plan cover all medical services, these company plans pay only for surgical operations.

The Bulletin comments on this situation as follows:

> A very serious point for us to consider is that by accepting these people and their terms we are allowing a very dangerous principle to be introduced into the practice of medicine: this is third party gain. It is a dangerous principle that a third party should make money out of the sick and squeeze out of both the sick and the doctors attending them profit which they have not earned. Our Medical Services Association exists, and we should urge people to join this. It gives infinitely more value for their money and is under close supervision and control.
>
> The modus operandi of these gentry is to send a curtailed check and, if the doctor protests, to utter all sorts of threats of blacklisting, etc., as well as to assure the doctor that every one else is accepting the cut. We think that the only answer to this sort of blackmail is to insist firmly on payment of our bill and ignore completely their threats. If we tell our patients the facts and show them where they can get equitable, honest and complete medical service on a health insurance basis, we shall have done them a service and helped to protect them, as well as ourselves, against what is fast becoming a racket.

PROGRAM OF INDIGENT CHILD CARE IN TEXAS

The Texas Department of Public Welfare, before entering on the program of aid to indigent children, conferred with the Council of Medical Economics of the State Medical Association of Texas. After frequent conferences and correspondence, the following agreement as to the medical services involved was drawn up and accepted by both parties:

> This plan for securing medical examinations of parents claiming physical or mental incapacity under the Aid to Dependent Children Program constitutes an agreement made by and between the Texas State Department of Public Welfare and the State Medical Association of Texas and is predicated on the following statement of facts:
>
> The Forty-Seventh State Legislature, acting under the authority of a constitutional amendment, appropriated $1,500,000 to use for aid to dependent children. The legislature also made provision for participation in federal funds made available through the Social Security Act for aid to dependent children, which means that Texas can receive $1,500,000 in federal funds for this program. In other words, the State Department of Public Welfare is now responsible for the administration of $3,000,000 per year to be used in paying grants to dependent children who meet certain eligibility requirements.
>
> The constitution of Texas, article III, section 51d, restricts payments of state funds to $8 per month for one child in an eligible family. If there are two or more children in one home, the grant from state funds is limited to $12 per month. This means that out of a total federal-state expenditure of $3,000,000, including $150,000 for administrative cost, one child may not receive a grant in excess of $16 per month. If there is more than one child in an eligible family, the maximum grant allowable per family for federal-state funds is $24 per month.
>
> To participate in the Aid to Dependent Children Program, the child must meet definite eligibility requirements. He must be under 14 years of age, a citizen of the United States, a resident of Texas, and be deprived of parental support or care by reason of death of parent, continued absence from home or physical or mental incapacity.
>
> The parent of every child claiming eligibility on the basis of physical or mental incapacity must have a medical examination to determine the presence and extent of incapacity. The parent must also be reexamined from time to time to establish continuing eligibility. As already pointed out, administrative funds are limited to $150,000 per year (5 per cent). The administrative fund must be used to pay salaries and travel of the investigators, clerical assistance, postage, physical and mental examinations, and so on.
>
> The department will pay $3 for routine physical or mental examinations, and the same fee ($3) will be allowed for each routine reexamination. The routine examination must include an examination of the entire body, a urinalysis, blood pressure, temperature and pulse rate. Only physicians who are legally licensed to practice medicine in Texas will be used, and the parent will have the choice of selecting from among these. Appropriate examination forms will be furnished by the State Department of Public Welfare, and the examination fee will be paid directly to the physician.
>
> The department realizes that in rare cases it may not be possible to determine physical or mental incapacity without more complete laboratory work, x-ray and consultation with a specialist. For these few cases the examining physician is requested to write an individual letter to the State Department of Public Welfare, Austin, Texas, setting forth in detail the amount of laboratory test, x-ray and consultation services required. The department will then work out, on an individual case basis, with the examining physician, fees to be paid for the required services in line with those received in private practice for similar services from persons in average financial condition in the community in which the examining physician resides.

OFFICIAL NOTES

ADDRESSES BY THE PRESIDENT-ELECT

Dr. Fred W. Rankin, President-Elect of the American Medical Association, has been scheduled to deliver the following addresses during January:

January 16-17.—Visiting Surgeons' Club, Cleveland.
January 21.—Bronx County Medical Society, New York.

Medical News

(PHYSICIANS WILL CONFER A FAVOR BY SENDING FOR THIS DEPARTMENT ITEMS OF NEWS OF MORE OR LESS GENERAL INTEREST: SUCH AS RELATE TO SOCIETY ACTIVITIES, NEW HOSPITALS, EDUCATION AND PUBLIC HEALTH.)

CALIFORNIA

Refresher Courses.—The University of California Medical School will offer a refresher course on "Clinical Aspects of New Therapy" in Toland Hall, University of California Hospital, San Francisco, January 5-7. Designed to meet the needs of practicing physicians, the course will include the following subjects: sulfonamide drugs, drugs used on the central nervous system, organotherapy, and drugs used in treatment of diseases of the adrenal glands; new drugs acting on the heart and circulation, and clinical aspects of nutrition. Members of the faculty will participate. Complete programs may be obtained from the Dean's Office, University of California Medical School, San Francisco.

Effects of Different Lunches on School Children.—The state defense nutrition committee, the department of public health and the University of California Medical School, San Francisco, are cooperating in a two year nutrition study among school children in Santa Barbara County, to begin early this year. The study will be made by evaluating the effects of different types of lunches on the nutritional status of children in a representative section of Santa Barbara County. Three groups of two hundred children each will receive three different types of lunch. One group will eat lunches they are accustomed to bring from home. A second group will eat a scientifically balanced lunch prepared at school, and the third group will eat lunches from home but will be given a supplementary pellet supercharged with vitamins and minerals. The nutritional status of all the children will be checked before the study begins; vitamin and mineral reserves will be measured. Medical examinations of the children will be made at regular intervals, and a final survey will be made of the results of the program.

GEORGIA

Society News.—Dr. Cyrus W. Strickler Sr., Atlanta, addressed the Muscogee County Medical Society recently in Columbus on "Development of Treatment Methods in the Management of the Pneumonias."——The Georgia Urological Association was addressed, October 9, among others, by Drs. William F. Reavis and Lovick W. Pierce, Waycross, on "Renal Calculi."

Hospital News.—A federal grant of $160,000 has been given to the Phoebe Putney Memorial Hospital, Albany, to provide construction and equipment of a new hospital and alterations. Existing facilities are inadequate at the hospital on account of increased defense activities at Turner Field, Darr Aero Tech Flying School and in the nearby towns of Bainbridge, Moultrie, Valdosta and Americus.

Annual Pediatric Meeting.—The Georgia Pediatric Society convened in annual session at the Biltmore Hotel, December 11. The speakers included:

Dr. Albert B. Sabin, Cincinnati, Epidemiology of Poliomyelitis.
Dr. Philip F. Barbour, Louisville, Ky., Active and Passive Immunization of Tetanus.
Dr. George M. Lyon, Huntington, W. Va., Sulfaguanidine Treatment of Bacillary Dysentery.

At an evening session Dr. Lyon discussed "Chemotherapy of Meningitis"; Dr. Barbour, "Rehabilitation of Poliomyelitis Patients," and Dr. Sabin, "Pathology, Symptomatology and Treatment of Poliomyelitis."

ILLINOIS

Precommitment Services for Mental Patients.—At a recent meeting of the managing officers of the mental institutions in Jacksonville, a plan of precommitment consultation service was adopted to be available to judges, physicians, relatives of patients, private and public welfare agencies and other professional groups. The service consists of an examination and an evaluation of a mental patient for whom admission to a state mental hospital is planned, to determine whether such institutionalization is needed and whether a more satisfactory plan can be worked out for the patient within his own community. The precommitment service will be limited to a single diagnostic interview, after which recommendations will be given the relatives, judge, physicians and other referring individual or organization. It will be offered both at the hospital itself and also on a regular monthly basis in several centers of population in the counties the hospital serves, according to the *Welfare Bulletin*.

Chicago

Courses in Obstetrics.—Five postgraduate courses in obstetrics, each of four weeks' duration, will be offered at the Chicago Lying-In Hospital between January 12 and June 6, under the sponsorship of the state department of health and the Children's Bureau, U. S. Department of Labor. Features of the program consist of observations on current managements of normal and abnormal states of the pregnant, parturient and puerperal patient. There will be lectures, demonstrations, clinics and other teaching means to augment the operating room and birth room observations, and ward round discourses. The courses will be run on a nonprofit basis. A deposit of $25 is required on registration, $10 of which will be refunded at the completion of the course. Additional information and application blanks may be obtained by request from the Postgraduate Course, Department of Obstetrics and Gynecology, 5848 Drexel Avenue.

MARYLAND

Mosquito Control Program.—The state department of health has set up a mosquito control program in Maryland to control mosquito breeding in defense areas, primarily in the vicinity of the Aberdeen Proving Ground, the Edgewood Arsenal, the Bethlehem Steel Company, the Glenn L. Martin Company, Camp Holabird and the U. S. Naval Powder Factory at Indian Head. The work will consist of the application of oil to sluggish streams, small ponds and stagnant pools, the clearing of underbrush adjacent thereto and the draining of salt marshes where necessary. The project will be under the direction of a sanitary engineer detailed by the U. S. Public Health Service and the state bureau of sanitary engineering. The WPA has allocated $42,000 to Maryland for the program and furnished all labor.

MASSACHUSETTS

Evans Memorial Lecture.—Dr. Alvah H. Gordon, Montreal, gave the first in the series of Robert Dawson Evans Memorial Lectures in Evans Auditorium, Boston, November 21. His subject was "Bone Changes in Certain Medical Diseases."

Personal.—Dr. John E. Gordon, Charles Wilder professor of medicine and epidemiology and head of the department, Harvard Medical School, and director of the American Red Cross-Harvard Hospital Unit in southwestern England, arrived in New York, November 10, on the Atlantic clipper. He came for a vacation and to recruit American staff members for the unit.

Society News.—Dr. John Romano, Boston, among others, addressed the Boston Society of Biologists, December 10, on "Psychiatric Manifestations of the Spontaneous Hypoglycemia."——The Massachusetts Society for Research in Psychiatry was addressed, December 9, by Saul Rosenzweig, Ph.D., and Dr. George E. Gardner, Boston, on "Sudden Death in the Anamnesis of Schizophrenia" and "Transient Psychotic Manifestations in Children."

Cancer Work at Harvard.—Harvard University and the Massachusetts General Hospital, Boston, have reached an agreement whereby the main phases of the work in cancer treatment and research now being carried on by the Collis P. Huntington Memorial Hospital of Harvard Medical School will be transferred to the general hospital. The step was taken because of the belief that the care and treatment of medical specialties can be more efficiently handled as part of a large institution of general scope than in smaller individual units. Under the new arrangement there will be transferred to the Massachusetts General Hospital the inpatient and outpatient services at the Huntington Memorial. The laboratory and research work now carried on by the Harvard Cancer Commission, established in 1899, will continue under the supervision of the commission. The research activities of Dr. Joseph C. Aub, associate professor of medicine at the Harvard Medical School, will be carried on at the general hospital in connection with the tumor clinic which the hospital has been conducting for many years. The remainder of the varied research work of the cancer commission will be continued in conjunction with the medical school. The equipment of the general hospital, including its 1,000,000 volt x-ray machine, will be available to all

patients at both the Huntington and the general hospital, and the Harvard supply of radium, heretofore used at the Huntington, will be available to the Massachusetts General Hospital. There will be no reduction in the number of beds for cancer patients in the community.

MICHIGAN

Society News.—The general practice section of the Wayne County Medical Society and the Detroit Pediatric Society were addressed, December 15, by Dr. Richard C. Eley, Boston, on "Nutritional Requirements of Infants and Children." Dr. Herrman L. Blumgart, Boston, will address a general meeting of the society, January 15, on "Coronary Collateral Circulation: Its Clinical and Pathological Significance."

Changes in Health Officers.—Dr. Frederick S. Leeder, Coldwater, director of the Branch County Health Department, has resigned to engage in research. Dr. Robert B. Harkness, Battle Creek, formerly director of the Barry County health unit, will serve as acting director of Branch County pending a permanent appointment.——Dr. Henry G. Steinmetz, Arlington, Va., is health commissioner of Genesee County during the absence of Dr. Leslie V. Burkett, Flint, who is taking postgraduate work in public health at Columbia University, New York.

Reelection of Examining Board.—The Michigan State Board of Registration in Medicine at its annual meeting in Lansing, October 14, reelected Drs. Elmer W. Schnoor, Grand Rapids, president; Claude R. Keyport, Grayling, vice president, and J. Earl McIntyre, Lansing, secretary. Attorney General Herbert J. Rushton appointed Mr. Milton G. Schancupp, Owosso, special assistant attorney general, as legal counsel and investigating officer to the board. During the week previous to the board's meeting the governor reappointed Drs. Schnoor and Luther Peck, Plymouth, as members of the board and named Dr. Eugene S. Thornton, Muskegon, to succeed Dr. Garner M. Byington, Detroit; Dr. Harold L. Morris, Detroit, to succeed Dr. William C. Ellet, Benton Harbor, and Dr. Charles W. Balser, Detroit, to succeed Dr. Francis B. Jarzembowski, Detroit. Dr. Morris and Dr. Thornton have previously served as members of the board.

MINNESOTA

Dr. Coller to Give Judd Lecture.—Dr. Frederick A. Coller, professor of surgery and chairman of the department of surgery, University of Michigan Medical School, Ann Arbor, will deliver the E. Starr Judd lecture at the University of Minnesota in the Chemistry Auditorium, Minneapolis, January 21. His subject will be "A Review of Studies on Water and Electrolyte Balance in Surgical Patients." The late Dr. Judd, an alumnus of the University of Minnesota Medical School, established this lectureship in surgery a few years before his death.

MISSOURI

Annual Hodgen Lecture.—Dr. Edwin P. Lehman, professor of surgery and gynecology at the University of Virginia Department of Medicine, Charlottesville, will deliver the annual Hodgen Lecture at the St. Louis Medical Society, January 13. His topic will be "The Use of Heparin."

Personal.—Dr. Evarts A. Graham, professor of surgery at Washington University Medical School, St. Louis, has been elected a member of the Royal Society of Sciences of Uppsala, Sweden, an organization founded in 1710. Dr. Graham was the only American among the five members of the society elected at this time. The election of membership is based on scientific merit. Dr. Graham is responsible for advances particularly in the fields of cholecystography and of chest surgery. ——Dr. Hugh W. Maxey has been appointed surgeon and directing head of the Missouri State Penitentiary Hospital, Jefferson City, newspapers report.

NEBRASKA

District Meetings.—The Fourth Councilor District Medical Society devoted its recent meeting to a symposium on fractures sponsored by the Six County Medical Society. The speakers were Drs. William L. Sucha, Omaha, James W. Martin, Omaha, and Charles F. Ferciot, Lincoln. ——The Seventh Councilor District Medical Society was addressed in Sutton recently by Drs. Joseph E. Uridil, Hastings, on "Infections of the Hand"; Raymond J. Wyrens, Omaha, on "Use of Sulfonamide Drugs in Internal Medicine," and Rolland Russell Best, Omaha, "Application of the Biliary Flush in Biliary Tract Disease."

NEW YORK

Distribution of Sulfadiazine for Pneumococcic Infections.—Sulfadiazine has recently been made available through the state department of health to registered doctors of medicine and hospitals in the state, exclusive of the city of New York, according to *Health News*. As with sulfapyridine and sulfathiazole, state issued sulfadiazine is intended only for those patients for whom purchase of this drug would be a hardship. The drug may be obtained in packages of sixty tablets of 0.5 Gm. each from laboratory supply stations. To comply with the ruling of the state board of pharmacy, the actual signature of physicians requesting sulfonamide drugs is required.

Personal.—Oscar Riddle, Ph.D., of the department of genetics of Carnegie Institution of Washington at Cold Spring Harbor, has been elected a foreign corresponding member of the Academia Nacional de Medicina, according to *Science*.—— Dr. William H. Ross, Brentwood, first president of the Suffolk County Tuberculosis and Public Health Association, was presented with a citation for public service in recognition of his "farsighted leadership and unselfish service." The presentation was made on the occasion of the association's twenty-first anniversary in Patchogue recently.——Dr. John R. Ross, superintendent of the Harlem Valley State Hospital, Wingdale, was appointed head of the Hudson River State Hospital at Poughkeepsie, effective November 30, succeeding the late Dr. Ralph P. Folsom. Dr. Ross has been in the state hospital service since 1908.

Postgraduate Courses.—A postgraduate course on general medicine, arranged by Dr. Walter W. Palmer, New York, for the Westchester County Medical Society, will open at New York Hospital, Westchester division, White Plains, January 14, with a talk on "Nephritis" by Dr. Dana W. Atchley, associate professor of medicine, Columbia University College of Physicians and Surgeons. On March 11 asthma will be discussed by Dr. Albert VanderVeer, New York, and on May 13 rheumatic fever by Dr. Homer F. Swift of the Rockefeller Institute for Medical Research. A course on general medicine was given before the Rockland County Medical Society at the Summit Park Sanatorium, Pomona. Speakers, all of New York, included:

Dr. David D. Moore, Diabetes Mellitus, November 7.
Dr. Ralph G. Stillman, Significance of Laboratory Tests and Methods in the Practice of Medicine, November 14.
Dr. John D. Lyttle, Nephritis, November 21.
Dr. Norman H. Plummer, Newer Chemotherapeutic Methods, November 28.
Dr. William B. Sherman, Asthma, December 5.
Dr. Leslie P. Barker, Syphilis, December 12.
Dr. Paul Reznikoff, Diagnosis and Treatment of Anemia, December 19.

New York City

Academy of Medicine Seeks Million Dollar Endowment.—The New York Academy of Medicine has launched a five year plan to obtain an endowment of "at least a million dollars" to insure an annual income sufficient to meet the requirements of continually enlarging services to the community, according to *New York Medical Week*.

Society Honors Ex-Presidents.—The first annual dinner to ex-presidents of the Medical Society of the County of Kings and the Academy of Medicine of Brooklyn will be held at the Hotel St. George, Brooklyn, January 17. Dr. Samuel J. Kopetzky, president of the state medical society, will present medals and scrolls, and speakers will be Hon. Fiorello H. LaGuardia, mayor of the city of New York, and Dr. Morris Fishbein, editor of THE JOURNAL. The Doctors' Orchestra will furnish the music.

Lectures for the Public.—The seventh series of lectures for the public on the army, art and romance of medicine, sponsored by the New York Academy of Medicine, opened on November 13 with the delivery of the Linsly R. Williams Memorial Lecture by Dr. James Alexander Miller, professor of clinical medicine, Columbia University College of Physicians and Surgeons. Dr. Miller's subject was "Tuberculosis: The Known and the Unknown." The second lecture was given by Dr. Tracy Jackson Putnam, professor of neurology and neurosurgery at Columbia, December 11, on "Mechanisms of the Mind." Other lectures in the series will be:

Dr. Abraham A. Brill, The Freudian Epoch (New York Academy of Medicine Anniversary Discourse), January 22.
Dr. Arnold L. Gesell, New Haven, Conn., Creative Behavior in Child and Adult, February 26.
Dr. Norman H. Jolliffe, History of Vitamin B, March 26.
Dr. Anton J. Carlson, Chicago, Newer Knowledge on Nutrition, April 23.

OHIO

Chief of Child Hygiene Division.—Dr. Susan P. Souther, formerly child hygiene physician in the bureau of child hygiene, Connecticut State Department of Health, Hartford, has been appointed chief of the division of child hygiene in the Ohio State Department of Health, Columbus.

Dr. Spies Wins Pharmaceutic Award.—The Award of Distinction of the American Pharmaceutical Manufacturers' Association was presented to Dr. Tom D. Spies, associate professor of medicine, University of Cincinnati College of Medicine, Cincinnati, during a meeting in Washington, D. C., December 8, in recognition of his work on nicotinic acid. The award is made annually to an investigator who, in the opinion of the committee, has made a fundamental contribution to public health in the field of drug therapy.

Society News.—Dr. Marion A. Blankenhorn, Cincinnati, discussed "Avitaminosis" before the Summit County Medical Society in Akron, December 2.——"Health in the Emergency" was discussed at a meeting of the Public Health Federation, Cincinnati, December 1, by Drs. Harry S. Sullivan, Washington, D. C., Arthur T. McCormack, Louisville, and John T. O'Rourke, D.D.S., Louisville.——Dr. Elmer L. Sevringhaus, Madison, Wis., discussed "Endocrine Treatment of Pituitary Disorders" before the Academy of Medicine of Cleveland, December 19. A tuberculosis committee has been formed by the academy to study the local tuberculosis setup and to make recommendations to the county commissioners; members of the committee are Drs. Joseph T. Wearn, Cleveland, chairman; Robert H. Browning, Warrenville; Raymond C. McKay, Edgar P. McNamee, Ulysses G. Mason Jr. and Charles F. Good, Cleveland.

Changes in the Faculty at Western Reserve.—Recent promotions on the faculty of Western Reserve University School of Medicine, Cleveland, include the following:

Dr. James R. Driver to associate clinical professor of dermatology and syphilology.
Donald E. Gregg, Ph.D., associate professor of physiology in the department of medicine.
Dr. Fred W. Dixon, assistant clinical professor of otolaryngology.
Dr. Curtis F. Garvin, assistant professor of medicine.
A. Sidney Harris, Ph.D., assistant professor of physiology.
Dr. Joseph Seifter, assistant professor of pharmacology.
Dr. Reginald A. Shipley, assistant professor of medicine.
Dr. Charles T. Way, assistant clinical professor of medicine.
Dr. Clarence C. R. Jackson, assistant clinical professor of otolaryngology.

New appointments to the school include Ralph I. Dorfman, Ph.D., as assistant professor of biochemistry, assigned to the Brush Foundation.

OREGON

Changes in Health Officers.—Dr. Harry J. Anderson, Corvallis, has been named health officer of Benton County, succeeding Dr. William T. Johnson, who held the position for many years. The latter, who is retiring, also acted as health officer of Corvallis.——Dr. Clarence R. E. Lindgren, Eugene, has been appointed health officer of Lane County.

Society News.—Dr. William W. P. Holt, Medford, discussed "Pylorospasm" before the Jackson County Medical Society in Ashland, November 12.——Dr. Joe B. Davis, Portland, addressed the Marion-Polk Medical Society in Salem, November 18, on disorders of the foot.——Officers of the state medical association and the Mid-Columbia Medical Society met jointly at Hood River, November 17; Dr. William W. Baum, Salem, president of the state association, discussed activities of the society and Dr. Roger H. Keane, Portland, postgraduate educational opportunities.——The Umatilla County Medical Society devoted its meeting in Pendleton, November 12, to a panel discussion on sulfonamide compounds; the speakers were Dr. Norman A. David, professor of pharmacology, University of Oregon Medical School, Portland, and Dr. Joseph P. Brennan, Pendleton.——Dr. Stuart W. Harrington, Rochester, Minn., discussed "Diagnosis and Treatment of Carcinoma of the Breast" before the North Pacific Surgical Association, Portland, November 10.

VIRGINIA

Personal.—Dr. Warren T. Vaughan, Richmond, was awarded the honorary degree of master of science by the University of Michigan, Ann Arbor, October 4.——Dr. Clifford E. Waller, Silver Spring, Md., formerly medical director U. S. Public Health Service, has been appointed health officer of the newly organized health department in Loudoun County at Leesburg.

Grants at Medical College of Virginia.—Dr. Everett I. Evans, instructor in pharmacology and surgery, Medical College of Virginia, Richmond, has been given a grant of $6,300 by the federal government, Office of Scientific Research Development, for further research on surgical shock, and Dr. John H. Scherer, assistant professor of medicine, Medical College of Virginia, Richmond, a grant of $3,000 from the John and Mary R. Markle Foundation to continue his work in reticulocytosis in jaundice.

ALASKA

New Medical Society in Kodiak.—The Medical Society of Fort Greely, Kodiak, was organized at a meeting of physicians of the Naval Air Station and the adjoining army post, November 21. Nurses and enlisted personnel of the medical departments on Kodiak Island will be privileged to attend the monthly meetings. Lieut. Harry E. Balch, Kodiak, was chosen first president of the society. The organization meeting of the new group, called by Major Hiram S. Yellen, formerly of Buffalo, followed the opening of a new wing at the Fort Greely Station Hospital. Major Yellen is chief of the hospital.

GENERAL

Commonwealth Fund Aids China.—The Commonwealth Fund, New York, has made a grant of $50,000 to the American Bureau for Medical Aid to China for the support of the Emergency Medical Service Training School in Kweiyang, China, according to *Science*.

National Social Hygiene Day.—The sixth National Social Hygiene Day will be observed throughout the country on February 4 with the theme "Keep America Strong, Help Build Better Health." Stress will be placed on the need for intensified action against syphilis and gonorrhea in industrial centers as well as military areas. Special attention will be directed toward the full realization of the menace of organized prostitution in areas adjacent to concentrations of armed forces and in industrial centers. Additional information concerning national and local programs may be received from the American Social Hygiene Association, Inc., 1790 Broadway, New York.

Annual Prize in Obstetrics.—The American Association of Obstetricians, Gynecologists and Abdominal Surgeons announces its annual "Foundation Prize." Three copies of all manuscripts and illustrations entered in a given year must be in hands of the secretary of the association before June 1. Manuscripts must be limited to five thousand words and be typewritten in double spacing on one side of the sheet. Illustrations should be limited to such as are required for a clear exposition of the thesis. A nom de plume must be used. The prize will be $150, and those eligible to compete include interns, residents or graduate students in obstetrics, gynecology or abdominal surgery and physicians who are actively practicing or teaching obstetrics, gynecology or abdominal surgery. Dr. James R. Bloss, 418 Eleventh Street, Huntington, W. Va., is secretary of the association.

Nutrition Fellowship at Pittsburgh University.—The first of the fellowships in nutrition recently offered by Swift & Company, Chicago, will assist a study on the nature of fats and oils at the University of Pittsburgh, Pittsburgh. The work is under the direction of Charles G. King, Ph.D., professor of chemistry, University of Pittsburgh, and Herbert E. Longenecker, Ph.D., Buhl Foundation research fellow and assistant professor of chemistry. Dr. King, who received his degree at University of Pittsburgh in 1923, is known for his discovery of the chemical identity of vitamin C. Karl F. Mattil, Ph.D., has been appointed research fellow under the Swift grant. All projects to be eligible for the new Swift grants should be aimed at development of fundamental information on the nutritive properties of foods or the application of such information to the improvement of American diet and health.

Vitamin Research Wins Chandler Medal.—Robert R. Williams, D.Sc., chemical director of the Bell Telephone Laboratories of New York, and Roger J. Williams, Ph.D., professor of chemistry, University of Texas, Austin, have been awarded the Charles Frederick Chandler Medal of Columbia University for their work in vitamin chemistry. The former was named for his work on vitamin B_1 and the latter for his discovery of pantothenic acid and for contributions to the knowledge of the vitamin B complex. In commenting on the prize the New York *Times* states that this is the first time since the establishment of the award in 1910 that two persons have been named as recipients. The prize is a gold medal awarded annually to the person appointed Chandler lecturer under the Charles Frederick Chandler Foundation established by alumni and former students of Professor Chandler.

Committee on Industrial Ophthalmology.—The Section on Ophthalmology of the American Medical Association and the American Academy of Ophthalmology and Otolaryngology have appointed a joint committee on industrial ophthalmology to engage in appraisals of industrial visual testing technics, instrumentation, interpretation of results, and to promote programs of education among workers, employers, the medical profession and others. Members of the committee representing the Section on Ophthalmology are Drs. Albert C. Snell, Rochester, N. Y., chairman; Arthur M. Culler, Dayton, Ohio, and Hedwig S. Kuhn, secretary, Hammond, Ind. Members for the academy are Drs. Thomas D. Allen, Chicago; Edwin B. Dunphy, Boston, and John B. Hitz, Milwaukee. Working and advisory relationships have been developed between the new committee and the Council on Industrial Health of the American Medical Association.

Dr. Hume to Head China Famine Relief.—Dr. Edward H. Hume, New York, director of the Christian Medical Council for Overseas Work and former president of the Colleges of Yale-in-China, has been made chairman of the China Famine Relief U. S. A. Inc. founded in 1928. Since 1938 the organization has been sending funds to the war stricken country through the Church Committee for China Relief, which it formed in conjunction with the Federal Council of the Churches of Christ in America and the Foreign Mission Conference of North America. The Church Committee is a member agency of United China Relief. Dr. Hume was born in India of American parents. He graduated at Johns Hopkins University School of Medicine, Baltimore, in 1901. From 1905 to 1927 he was associated with the Colleges of Yale-in-China at Changsha, serving as president from 1923 to 1927. Returning to America, he became executive vice president of the New York Post-Graduate Medical School and Hospital, a position he held until 1933. He became director of the Christian Medical Council for Overseas Work in 1938.

War Conditions and Tuberculosis.—War conditions and the lengthened hours of work in defense industries have reversed the downward trend of tuberculosis, the New York *Times* stated, November 29, in commenting on a survey in the United States and abroad made by Godias J. Drolet, assistant director and statistician of the New York Tuberculosis and Health Association. The survey shows that already a decided increase in tuberculosis mortality is taking place in Europe. Although in the United States generally there has been no sharp change in the rate of this disease, danger signals are appearing, it was stated. In forty-six large American cities surveyed, nineteen showed a rise in tuberculosis mortality during the period from January 1 to November 15. For the entire nation the death rate was 45.9 per hundred thousand of population against 47.1 the year before. In the large cities, however, the death rate was 26 per cent above that of the nation at large. San Antonio had the highest rate, 144 per hundred thousand of population, while St. Paul, with only twenty-eight deaths, had the lowest. The rate in the five cities with more than one million population was for Philadelphia 65, Chicago 61, New York 54, Detroit 51 and Los Angeles 50. Mr. Drolet, who made a tuberculosis survey for the Rockefeller Foundation during the World War, pointed out that with increased activity and overtime work it is urgent that there be a corresponding increase in our vigilance against tuberculosis.

Tracing Source of Typhoid Outbreak.—An outbreak of typhoid in England was recently traced to a carrier, according to a report of Dr. John E. Gordon, director of the American Red Cross-Harvard Field Hospital in England. On September 4 and 9 two small boys living within 75 feet of each other were admitted to the Chippenham Isolation Hospital with a diagnosis of typhoid. On September 15 the Harvard unit was asked to investigate. A search for typhoid carriers and Widal reactions was made in every member of both families and in the families of three food merchants in the vicinity common to the two families. No infected person was discovered. Other possible sources such as milk, drinking water, vegetable grocers and places visited were eliminated. On October 3 the two children had recovered so that they could converse and admitted having a common playmate, although they did not play with each other. A visit to the home of the third child revealed that the three children frequently played in the river, actually a small creek flowing within 30 feet of the home of this last child. This proved to be the site of a broken sewer which often overflowed and which had overflowed during the critical incubation period of the two infected children. No case of the disease had been reported in the community for ten years. Subsequent testing disclosed the mother of the third child to be a typhoid carrier and one of a new group of evacuees in the village. It was believed that the children exchanged cookies or bread and butter and that the disease spread through the food prepared by the carrier for her child. The laboratory tests showed that the carrier strain of the mother was phage type A, which was the strain found in the two sick children.

CANADA

Faculty Changes at McGill.—Dr. Theodore R. Waugh, assistant professor of pathology, McGill University Faculty of Medicine, Montreal, has been made associate professor of pathology, and Dr. William L. Ritchie was promoted to be associate professor of radiology and head of the department. Dr. Carleton B. Peirce, director, department of radiology and radiologist in chief at the Royal Victoria Hospital, was made associate professor of radiology.

Lectures for General Practitioners.—The Academy of Medicine of Toronto announces a special course of lectures for the general practitioner, January 5-9. The program will include the following:

Dr. Percy A. Sarjeant, The Hazard of Air Raids, January 5.
Dr. John Harold Couch, Personal Protection in Air Raids, January 5.
Dr. Robert W. Wesley, Significance of Uterine Bleeding from Non-malignant Causes, January 6.
Dr. Nicholas D'Arcy Frawley, Management of Toxemia of Pregnancy, January 6.
Dr. Hamnett A. Dixon, Diagnosis and Treatment of Common Skin Lesions, January 8.
Dr. Ross A. Jamieson, Heart Disease in General Practice, January 8.
Dr. Frank Burns Plewes, Treatment of Hemorrhoids and Varicose Veins, January 9.
Dr. Charles R. B. Crompton, Significance and Danger of Painless Hematuria, January 9.

There will be a panel discussion on the sulfonamide derivatives, January 7, by Drs. Wilbert Hurst Brown, Philip H. Greey, Wilfred K. Welsh, James L. McCollum and Leslie Nelles Silverthorne. All are from Toronto.

LATIN AMERICA

Brazilian Quarterly Becomes Official Journal for Ophthalmologic Congress.—*Ophthalmologica Ibero-Americana*, the Brazilian quarterly abstract journal, is now the official organ of the Pan American Congress of Ophthalmology. *Ophthalmologica* has appeared only in Portuguese, but after January 1 all abstracts will be printed in Spanish and English or in Portuguese and English. Original articles, reports of scientific activities of the congress, news items and so on will be reproduced in all three languages. Dr. Moacyr E. Alvaro, São Paulo, Brazil, is managing editor, and the committee on literary exchange of the congress, consisting of all the ophthalmic editors of the Western Hemisphere under the chairmanship of Dr. J. Lyó Pavia, Buenos Aires, Argentina, forms the editorial board. *Ophthalmologica Ibero-Americana* will be sent without charge to all members of the Pan American Congress of Ophthalmology who shall have paid dues in advance. An annual membership fee of $5 will be sufficient to supply the journal to all members and to finance the second Pan American Congress of Ophthalmology, which is to be held in Montevideo in November 1943. Interested ophthalmologists are asked to send their name and address together with the membership fee of $5 to Dr. Conrad Berens, secretary-treasurer of the Pan American Congress of Ophthalmology, 411 First Avenue, New York.

FOREIGN

Paderewski Hospital Opened.—The Paderewski Hospital at Edinburgh, Scotland, has been opened, newspapers reported, December 7. The hospital has been established in connection with a medical school as part of the Polish Medical Center, Edinburgh, which has been in operation since March. A memorial to the late Ignace Jan Paderewski, pianist and Polish premier, the hospital will be open to Polish civilian refugees, to Polish soldiers in Scotland and, in an emergency, to the British public. The city of Edinburgh gave the building, and funds from the Paderewski Testimonial Fund in America have assisted in the expenses. The fund was started before Paderewski's death as a tribute on the occasion of the fiftieth anniversary of his American debut. Newspapers stated that, once Poland is free, the hospital will be transferred there.

Foreign Letters

LONDON

(From Our Regular Correspondent)

Nov. 15, 1941.

The Mass Psychology Which Menaces the World

When Wilfred Trotter died in 1939 he was regarded as our greatest surgeon. But he had another title to fame; he was a philosopher. In 1908-1909 he contributed to the *Sociological Review* two essays on herd instinct, in which he questioned the accepted distinction between sociology and psychology. He argued that, as man was unknown as a solitary animal, every one must present the characteristics of a social animal or herd. He first dealt with instinct, which he defined as "inherited modes of reaction to bodily need or external stimulus." He distinguished three primary instincts: self preservation, nutrition and sex. But in gregarious animals, including man, he found a fourth instinct which modified and controlled these primary instincts. This he called the herd instinct. It was of three types: the aggressive, shown in the wolf and dog, the protective, shown in the sheep and deer, and the socialized, shown in the bee and ant. In the last the individual is completely absorbed into the major unit. Trotter held that the socialized type was the goal of man's development. Toward it his constantly growing altruism was directed. He expanded his essays into a famous book, which he published in 1916, entitled "Instincts of the Herd in Peace and War." As the extension of the original title shows, much that he wrote was inspired by the great war, then in progress. He gave to herd instinct a new importance in human psychology. He found in it the source of the individual's "opinions, credulities, disbeliefs and weaknesses." He wrote "Each one of us in his opinions, conduct, dress, amusement, religion and politics is compelled to be one of a class—the herd within the herd." Modern nations conformed to the herd types of the lower animals. Germany was "a perfect copy" of the wolf pack. "I am not intending to use a vague analogy," he said, "when I compare German society to the wolf, but to call attention to a real and gross identity. The psychic necessity which makes the wolf brave in a massed attack makes the German brave in a massed attack; the psychic necessity which makes the dog submit to his master's whip and profit by it makes the German submit to the lash of his officer and profit by it. . . . Such a people have no conception of the benign use of power but must regard war as an end in itself and peace as a somewhat irksome preparation for war."

All this was written in Trotter's book published twenty-five years ago. Today the *Times* gives, in its series of quotations under the caption "Old and True," the following extract from the book: "I admit to myself, quite frankly, my innermost conviction that the destruction of the German Empire is an indispensable preliminary to the making of a civilization tolerable by rational beings." This warning had no effect on our politicians, who would never dream that a surgeon, however eminent, or a philosopher, however acute, could provide any instruction of practical value on a political subject. A similar warning of the German peril given by a few public men, who included another philosopher, Frederick Harrison, the positivist, was ignored before the last great war. But now, as then, our enemies will find that they have made a mistake in relying on our normal aversion to militarism. If such warnings had been taken to heart how much suffering the world would have been saved! Trotter was by no means the only one among our public men who foresaw the danger, but no one dealt with it so scientifically and showed that it necessarily arose from the mass psychology of the Germans.

Is Alcohol in Any Sense a Food?

To a manual of scientific temperance teaching for the use of teachers and students, published by the Temperance Collegiate Association, Sir Frederick Gowland Hopkins, F.R.S., has contributed an essay on the relation of alcoholic beverages to nutrition. It is generally taught that as alcohol undergoes combustion in the body it acts as food. But Hopkins gives strong reasons for doubting this. Unlike our indispensable foods, alcohol disappears from the body in a few hours and cannot be stored. Its oxidation does not prove that it fulfils the functions of a food. Many deleterious substances are oxidized in the body and then eliminated. The energy liberated by the combustion of alcohol may well be unavailable for the support of any bodily function. Modern studies of the chemical events involved in muscular contraction give good reasons for doubting that alcohol can play a significant part in them.

These a priori considerations are supported by recent direct experiments which show that during work the oxidation of alcohol taken proceeds no faster than when the individual is completely at rest. Therefore the alcohol cannot have supplied energy for the work. In two young men whose diet and activities were carefully controlled, the rate of disappearance of alcohol from the body was studied for some hours when they were completely at rest. This rate was essentially the same on different occasions. Then the rate of disappearance was followed during bicycle riding, slowly for long periods or fast for shorter ones. The work did not increase the rate of disappearance of alcohol. It therefore could not have provided energy for the work done. The same result was obtained when the work was walking 20 miles over stiff country and in experiments on animals. The constancy of rate of combustion seems to be a device for protection rather than for utilization.

Even when no muscular work is done, heat derived from the combustion of food is necessary for maintaining the temperature of the body and the work of the internal organs. When the body is exposed to a low external temperature, combustion must increase to replace the heat lost through the skin. Can the combustion of alcohol supply any of this? Again, it was found that the combustion of alcohol was not quickened and so cannot meet the demand for greater heat production. Moreover, the dilating action on the cutaneous blood vessels increases the loss of heat. Therefore alcohol cannot protect against cold. Yet these two needs are just those which our non-nitrogenous foods characteristically supply. To suppose that alcohol can contribute in any direct sense to tissue growth would be absurd.

Insurance to Meet Hospital Bills

To encourage the formation of mutual insurance schemes which will assist the middle and professional classes to meet expenditure necessitated by illness necessitating specialist or surgical treatment undertaken in hospital pay beds or nursing homes, Lord Nuffield, famous for his munificent gifts to hospitals and medical education, has created a provident guaranty fund of $750,000. This will be available for the purpose of guaranteeing the financial solvency of provident schemes. A condition is that the schemes shall be approved by the trustees of the fund and provide substantial cover for subscribers and members of their families on a contributory basis. Lord Nuffield's fund is available for areas outside London. In the London area King Edward's Hospital Fund has appointed a committee, under the chairmanship of Sir Bernard Docker, to sponsor a provident scheme to cater for the requirements of the middle and professional classes.

The Supply of Milk to School Children

More than 2,700,000 children obtain milk regularly in the public elementary schools. Nearly 600,000 are served twice daily with a third of a pint; the remainder get it once daily. Some 350,000 get the milk free; the rest pay 1 cent per third of a pint, which is only a third of the price paid by ordinary consumers at present. A demand that the scheme should be extended to other schools has been made, as the parents who prefer these schools are now paying as taxpayers for other people's children to have more milk than their own. The Ministry of Food would like to see the scheme extended, but financial considerations stand in the way. While the hardship is admitted, it is pointed out that the public elementary schools are open to all.

BUENOS AIRES

(From Our Regular Correspondent)

Sept. 15, 1941.

Center for Breast Milk in Buenos Aires

The Breast Milk Center of Buenos Aires is a public institute for the collection, storage and distribution of breast milk on a noncommercial basis. It has the same aims as have similar institutes in other countries and differs from the breast milk collecting depots which were established in Argentina about twenty years ago in that only the superfluous breast milk is collected; that is, the center is supplied not merely by wet-nurses. The women are periodically subjected to clinical, roentgenologic and serologic examination. The mother's own child is not deprived of milk as is sometimes the case in wetnurses. This institute is staffed with social workers, who keep in contact with the families of the donors as well as of the recipients and take interest also in problems of clothing and diet and if necessary secure financial and legal aid, hospitalization for mother and child, vaccination against smallpox and diphtheria and so on. The institute has demonstrated its value in the twelve years of its existence. In 1940 milk obtained from 346 women was distributed to 1,237 children. Of 5,300 liters collected during 1940 about 2,800 liters could be dispensed without charge. Depending on the economic conditions, the price per liter varies from nothing to 8 Argentine pesos ($2.40).

Leprosy

According to E. M. Riveros, cases of leprosy have been observed in Colombia as follows: in 1936, 797 cases detected among 9,798 persons examined; in 1937, 834 among 31,279; in 1938, 767 among 31,268; in 1939, 654 among 55,633. The Asilo de Guadalupe, which admits healthy daughters of leprosy patients and has facilities for 250, was destroyed by fire in 1938 and its restoration has not been completed. San Bernardo can admit 400 children and 40 infants, another one 350 children and still another 100 children and 45 newborn infants. On May 1, 1940 these different institutes harbored 1,013 children. The state grants large subsidies to these institutions. The state has also had removed a large number of healthy children from the leprosariums; with financial support from the state they will remain under medical control up to the age of 15 years. From July 1938 to May 1940, 908 healthy children have been removed from the leprosariums; 532 were placed into the aforementioned asylums and 376 in families.

An analysis of the leprosy problem in the eastern provinces of Peru by M. H. Kuczynski-Godar and V. M. Pinedo demonstrates that the methods employed are inadequate. A well organized and medically well equipped rural colony is probably preferable to a hospital. In a first and cursory study of a district, 1.5 per cent of the population were found to have leprosy (44 cases).

According to Prof. V. Boettner (*Revista médica del Paraguay*, 1940) the state leprosarium of Paraguay at present has about 180 patients. Among the 140,000 patients admitted to the hospitals of Asunción from 1932 to 1936 there were 56 patients with leprosy. The number of leprous patients in the army during the three year Chaco war was 78. The medical examination of the mobilized men during the war disclosed in all 187 cases of leprosy. Paraguay has no districts with a great concentration of leprosy. Boettner was able to detect 30 cases in his private practice.

The High Incidence of Circulatory Diseases

The Departamento Nacional de Higiene of Argentina reports a high incidence of cardiac and vascular diseases, which has been observed also in other countries. In Buenos Aires, for instance, with a population of 2.4 millions and a total mortality of 26,535, the fatalities from circulatory diseases were 26.9 per 10,000 inhabitants, or 23 per cent of the fatalities. These diseases are more frequent among men than among women, the ratio being 2 to 1; at the age level above 60 the ratio between the two sexes becomes equalized.

The report states further that in the United States the mortality from circulatory diseases is relatively lower than in Argentina and, in spite of this they present an economic burden of $250,000,000, which is equivalent to 1,000 million Argentine pesos. The Departamento Nacional de Higiene estimates the burden for Argentina at 100 million Argentine pesos. The losses sustained by the pension fund owing to circulatory diseases go so far that of 514 persons who in 1940 wanted to take advantage of the pension funds 38.6 per cent intended to retire on account of circulatory diseases. The department attempts to remedy this problem by prophylactic measures. The large group of governmental employees is to be considered first. It has been suggested that, with the examination for tuberculosis, to which all governmental employees are to be subjected at five year intervals, an examination for circulatory diseases, especially for persons over 40, be combined. Persons in whom circulatory defects are discovered are, if possible, to be given other work more compatible with their state of health.

Personal

Guy Amerongen, a French industrialist who established a prize for the study of the interrelations between canned food and carcinogenesis but lost all of his possessions, is also seeking a new home in Argentina. The prize of 100,000 francs could not be distributed for twelve years. In 1939 the prize was awarded to Professor Roffo in recognition of a scientific contribution but was used by him to further the cultural relations between Argentina and France.

Dr. Mario Soto was appointed professor of pharmacology at the University of Buenos Aires. He will devote all his time to pharmacologic research and give up his clinical connections.

Prof. Aloysio de Castro, president of the academy of medicine in Rio de Janeiro and director of the general policlinic, has been given a doctorate honoris causa by the faculty of medicine in Buenos Aires.

Marriages

HASKELL WRIGHT FOX to Miss Ruby Lee, both of Greeneville, Tenn., in Asheville, N. C., Oct. 16, 1941.

ELDON EDGAR SMITH, Canton, Ohio, to Miss Patricia Matthews at Ludington, Mich., in October 1941.

PAUL D. ZUBRITZKY, McKees Rocks, Pa., to Miss Ilaria Sterniuk of Plymouth, Sept. 23, 1941.

FREDERICK H. BOWEN to Miss Henrietta Baldwin, both of Jacksonville, Fla., Oct. 25, 1941.

CLARENCE E. UMPHREY to Miss Helen Isabel Trimm, both of Detroit, Nov. 1, 1941.

JOSEPH CLEMENT FLYNN, Cincinnati, to Miss Deirdre Driver of Tampa, Fla., recently.

Deaths

Austin Smith McKitrick ● Elyria, Ohio; Eclectic Medical Institute, Cincinnati, 1888; University of Wooster Medical Department, Cleveland, 1902; fellow of the American College of Surgeons; past president of the Hardin County Medical Society and the Lorain County Medical Society; for many years member of the board of education of Kenton, Ohio; a founder of and surgeon to a hospital bearing his name; formerly trustee of the Ohio Northern University, Ada; aged 78; surgeon to the San Antonio Hospital, Kenton, and the Memorial Hospital, where he died, Nov. 3, 1941, of pneumonia.

Ellen Smith Stadtmuller, San Francisco; University of California Medical Department, San Francisco, 1912; member of the California Medical Association and the American Academy of Pediatrics; clinical instructor in pediatrics at his alma mater; chief of child hygiene, state department of public health; assistant chief, communicable disease department, Children's Hospital from 1918 to 1922; instructor and medical inspector, State Teachers College, from 1920 to 1922; member of the advisory committee of the National Society for the Prevention of Blindness; aged 58; died, Nov. 25, 1941.

Edwin Forrest Yancey, Sedalia, Mo.; Missouri Medical College, St. Louis, 1882; member and from 1910 to 1920 vice president of the Missouri State Medical Association; fellow of the American College of Surgeons; formerly medical director of the Missouri-Kansas-Texas Railroad Company; formerly chief of staff of the John H. Bothwell Memorial Hospital; aged 83; died, Nov. 21, 1941, of carcinoma of the prostate.

William Michael Mehl ● Buffalo; University of Buffalo School of Medicine, 1904; chairman of the New York State Commission for the Blind; consulting ophthalmologist for the state department of labor; on the staffs of the J. N. Adam Memorial Hospital, Perrysburg, and the Charitable Eye, Ear, Nose and Throat Hospital; aged 64; died, Nov. 28, 1941, in the Buffalo General Hospital.

Roy Thomas Rodaway, Roanoke, Ill.; Bennett Medical College, Chicago, 1913; member of the Illinois State Medical Society; past president of the Woodford County Medical Society; on the staff of St. Francis Hospital, Peoria; served during the World War; county coroner; aged 54; died, Nov. 4, 1941, at his summer home in South Haven, Mich., of coronary disease.

Francis LeRoy Phillips ● Assistant Surgeon, Lieutenant (junior grade) United States Navy, Mare Island, Calif.; University of Southern California School of Medicine, Los Angeles, 1936; entered the navy in August 1936; on the staff of the United States Naval Hospital; aged 32; died, Nov. 17, 1941, in the United States Naval Hospital, Brooklyn.

Theodore Henry Lemmerz, Jersey City, N. J.; New York Homeopathic Medical College and Hospital, New York, 1896; fellow of the American College of Surgeons; member of the Medical Society of New Jersey; ophthalmologist and director of clinics, Christ Hospital; aged 71; died, Nov. 8, 1941, of carcinoma of the rectum.

William Henry Marsh, Solomons, Md.; University of Maryland School of Medicine, Baltimore, 1876; member of the Medical and Chirurgical Faculty of Maryland; formerly associated with the U. S. Public Health Service; aged 90; died, Nov. 12, 1941, in the United States Marine Hospital, Baltimore, of carcinoma of the lung.

Thomas Douglass ● Ozark, Ark.; Missouri Medical College, St. Louis, 1889; president of the Tenth Councilor District Medical Society and secretary of the Franklin County Medical Society; for many years chairman for the Red Cross in Franklin County; aged 73; died, Nov. 7, 1941, in a hospital at Fort Smith.

Albert Clinton Leach, Orange, Mass.; Dartmouth Medical School, Hanover, N. H., 1894; member of the Massachusetts Medical Society; member of the local board of health for many years; aged 70; died, Nov. 9, 1941, in the Farren Memorial Hospital, Montague City, of cerebral hemorrhage.

William Herbert Mason ● Murray, Ky.; Vanderbilt University School of Medicine, Nashville, Tenn., 1899; past president of the Calloway County Medical Society; aged 66; medical director of a hospital bearing his name, where he died, Nov. 23, 1941, of Hodgkin's disease.

Ernest Maxwell Vardon, Los Angeles; University of Michigan Department of Medicine and Surgery, Ann Arbor, 1898; member of the California Medical Association; on the staff of the Queen of Angels Hospital; aged 71; died, Oct. 8, 1941, of retroperitoneal hemorrhage.

Douglas Hursh Vastine, Catawissa, Pa.; University of Pennsylvania School of Medicine, Philadelphia, 1923; member of the Medical Society of the State of Pennsylvania; aged 42; died, Nov. 14, 1941, in the Bloomsburg (Pa.) Hospital, of pneumonia and meningitis.

Robert Walker Hale, Thermopolis, Wyo.; Missouri Medical College, St. Louis, 1893; member of the Wyoming State Medical Society; formerly state senator and postmaster; at one time member of the state board of health; aged 72; died, Oct. 29, 1941.

Jim Hilliard Camp ● Pecos, Texas; University of Texas School of Medicine, Galveston, 1927; at one time instructor in anatomy at his alma mater; medical director of a hospital bearing his name; aged 39; was accidentally shot and killed, Nov. 16, 1941.

David Henry Davison, New York; College of Physicians and Surgeons, medical department of Columbia College, New York, 1876; member of the Medical Society of the State of New York; aged 87; died, Nov. 22, 1941, in the Mount Sinai Hospital.

William August Kriesel, Little Rock, Ark.; Minneapolis College of Physicians and Surgeons, 1897; member of the Arkansas Medical Society; served during the World War; on the staff of the Missouri Pacific Hospital; aged 71; died, Oct. 30, 1941.

Allen Johnson, San Antonio, Texas; Tulane University of Louisiana School of Medicine, New Orleans, 1916; member of the State Medical Association of Texas; served during the World War; aged 51; died, Oct. 13, 1941, of cerebral hemorrhage.

Mavity J. Spencer, Indianapolis; Central College of Physicians and Surgeons, Indianapolis, 1896; member of the Indiana State Medical Association; past president of the city board of health; formerly police surgeon; aged 70; died, Nov. 27, 1941, of coronary embolism.

Harry Theodale Rosenthal, New York; University and Bellevue Hospital Medical College, New York, 1911; member of the Medical Society of the State of New York; on the staff of the Bronx Hospital; aged 54; died, Nov. 23, 1941, of coronary thrombosis.

Lloyd Henry Sarchet, Wellington, Kan.; State University of Iowa College of Homeopathic Medicine, Iowa City, 1898; member of the Kansas Medical Society, city and county health officer; aged 69; died, Nov. 14, 1941, in St. Luke's Hospital.

Edgar Burton Probasco ● Glens Falls, N. Y.; Columbia University College of Physicians and Surgeons, New York, 1898; fellow of the American College of Surgeons; attending surgeon Glens Falls Hospital; aged 71; died, Nov. 5, 1941.

William S. Prather, Americus, Ga.; University of Georgia Medical Department, Augusta, 1889; member of the Medical Association of Georgia; formerly owner of the Americus Hospital; aged 73; died, Nov. 12, 1941, of coronary thrombosis.

Jesse Grim, Los Angeles; Western Pennsylvania Medical College, Pittsburgh, 1904; served during the World War; formerly passed assistant surgeon in the United States Public Health Service reserve; aged 64; died, Nov. 20, 1941.

Frank R. Smiley, Boonville, Mo.; Beaumont Hospital Medical College, St. Louis, 1888; member of the Missouri State Medical Association; was a member of the board of education and county coroner; aged 81; died in November 1941.

Leon Charles Lewis, Kansas City, Mo.; University Medical College of Kansas City, Mo., 1901; member of the Missouri State Medical Association; served during the Spanish-American and World wars; aged 65; died, Nov. 10, 1941.

Howe R. Coleman Sr., Collierstown, Va.; Medical College of Virginia, Richmond, 1898; chairman of the county board of health and formerly chairman of the county school board; aged 69; died, Nov. 7, 1941, of coronary thrombosis.

Clarence Atwood Rose, Ardmore, Pa.; Jefferson Medical College, Philadelphia, 1913; member of the Medical Society of the State of Pennsylvania; on the staff of the Bryn Mawr (Pa.) Hospital; aged 61; died, Nov. 13, 1941.

Edmund A. Terrell, Fredericks Hall, Va.; Medical College of Virginia, Richmond, 1885; member of the Medical Society of Virginia; for many years surgeon for the Chesapeake and Ohio Railway; aged 81; died, Nov. 18, 1941, of arteriosclerosis.

Mace Anderson Losee, Livingstonville, N. Y.; Cornell University Medical College, New York, 1902; member of the

Medical Society of the State of New York; for many years health officer; aged 63; died, Nov. 9, 1941.

Walker L. Stumbo, Lackey, Ky.; University of Louisville Medical Department, 1907; member of the Kentucky State Medical Association; county sheriff; formerly county judge; aged 58; died, Nov. 13, 1941, in Martin.

David J. Conyers, Halls, Tenn.; Memphis (Tenn.) Hospital Medical College, 1897; bank president; aged 76; died, Nov. 22, 1941, in the John Gaston Hospital, Memphis, of injuries received in an automobile accident.

Warren Lee Hall, Nicholls, Ga.; University of Georgia Medical Department, Augusta, 1913; member of the Medical Association of Georgia; served during the World War; aged 52; died in November 1941.

Alfred Martin Sutton, San Diego, Calif.; University of London Faculty of Medicine, London, England, and M.R.C.S., England, 1885; aged 82; died, Oct. 8, 1941, of fractured right femur and arteriosclerosis.

Eugene Le Roy Kellum, Richmond, Va.; University of Pennsylvania School of Medicine, Philadelphia, 1924; on the staff of the Grace Hospital; aged 43; was killed, Nov. 15, 1941, in an automobile accident.

Earl Hudson Turner ● Pampa, Texas; University of Texas School of Medicine, Galveston, 1934; aged 31; died, Oct. 12, 1941, as a result of injuries received in an accidental fall down an elevator shaft.

Baxter Rittenberry, Selma, Ala.; Birmingham Medical College, 1899; past president of the Dallas County Medical Society; aged 68; died, Nov. 19, 1941, of arteriosclerosis and cerebral hemorrhage.

Joseph Michel Arthur Valois, Montreal, Que., Canada; School of Medicine and Surgery of Montreal, Faculty of Medicine of the University of Laval at Montreal, 1900; aged 66; died, Nov. 16, 1941.

Mitchell Lawrence Moran, St. Petersburg, Fla.; Georgetown University School of Medicine, Washington, D. C., 1935; member of the Florida Medical Association; aged 31; died, Nov. 22, 1941.

Charles Henry Wilson, New Brighton, Pa.; Western Pennsylvania Medical College, Pittsburgh, 1906; aged 69; died, Nov. 1, 1941, in the Beaver Valley Hospital of carcinoma of the stomach.

Harvy Nally, Cainsville, Mo.; University of Missouri School of Medicine, Columbia, 1876; member of the Missouri State Medical Association; aged 86; died, Nov. 3, 1941, of coronary thrombosis.

Eugene Leffler Gilmore, Tallapoosa, Ga.; Marion-Sims College of Medicine, St. Louis, 1892; member of the Medical Association of Georgia; aged 72; died, Nov. 16, 1941, of septicemia.

K. Arvid Enlind, New York; College of Physicians and Surgeons, Baltimore, 1893; served during the World War; aged 68; died, Nov. 17, 1941, in the Veterans Administration Facility.

Robert Graham Contrell, New York; University of Pennsylvania Department of Medicine, Philadelphia, 1896; aged 68; died, Nov. 26, 1941, in Englewood, N. J., of carcinoma of the prostate.

Arthur Horton Mann ● Silver City, N. M.; University of Pennsylvania School of Medicine, Philadelphia, 1919; aged 48; died, Nov. 2, 1941, of lobar pneumonia and pulmonary tuberculosis.

James Stanley Gardner, Winnipeg, Man., Canada; University of Manitoba Faculty of Medicine, Winnipeg, 1923; aged 44; died, Nov. 25, 1941, of herpes zoster and pulmonary tuberculosis.

Calvin H. Maust, Lecompton, Kan.; Central Medical College of St. Joseph, Mo., 1905; member of the Kansas Medical Society; aged 65; died, Nov. 4, 1941, in a hospital at Topeka.

David H. Thornton, Princeton, W. Va.; College of Physicians and Surgeons, Baltimore, 1893; aged 76; died, Nov. 19, 1941, in Roanoke, Va., of coronary sclerosis and hypertension.

Paul Alexander Johnstone ● Kansas City, Mo.; University Medical College of Kansas City, Mo., 1903; aged 75; died, Nov. 9, 1941, of coronary occlusion and pulmonary embolism.

Hartwell Blount Hyde, Nashville, Tenn.; Vanderbilt University School of Medicine, Nashville, 1878; aged 88; died, Nov. 12, 1941, in the Davidson County Hospital of senility.

John William Smart, Cottontown, Tenn.; University of Tennessee Medical Department, Nashville, 1886; aged 80; died, Nov. 16, 1941, in the Protestant Hospital at Nashville.

Joseph M. Hale, Dearborn, Mo.; Ensworth Medical College, St. Joseph, 1891; member of the Missouri State Medical Association; aged 77; died, Nov. 10, 1941, in Kansas City.

Lawrence Miles Gwinn ● Denver; University of Colorado School of Medicine, Denver, 1913; on the staff of the Presbyterian Hospital; aged 53; died, Nov. 30, 1941.

Richard Churchill Travis ● Indianapolis; University of Michigan Medical School, Ann Arbor, 1925; served during the World War; aged 42; hanged himself, Nov. 23, 1941.

John Dana Robbins, Mount Ida, Ark.; Memphis (Tenn.) Hospital Medical College, 1902; member of the Arkansas Medical Society; aged 68; died, Oct. 29, 1941.

Charles Henry Walter, Oakland, Calif.; Hahnemann Medical College and Hospital of Philadelphia, 1894; served during the World War; aged 74; died, Nov. 22, 1941.

Milton Elliott Cannon, Riceville, Tenn.; Lincoln Memorial University Medical Department, Knoxville, 1910; served during the World War; died, Nov. 28, 1041.

Charles J. Ross, Dover, Ark.; Memphis (Tenn.) Hospital Medical College, 1892; past president of the Pope County Medical Society; aged 74; died, Nov. 15, 1941.

George McGowan Stuart, Cupar, Sask., Canada; Manitoba Medical College, Winnipeg, Man., 1906; chairman of the public school board; aged 59; died, Oct. 28, 1941.

George Lewis Fuquay, Coats, N. C.; North Carolina Medical College, Charlotte, 1916; served during the World War; aged 49; died, Nov. 28, 1941.

Henry M. Kerzman, Detroit; Universität Bern Medizinische Fakultät, Switzerland, 1921; aged 53; died, Nov. 22, 1941, in the Harper Hospital.

Frederick David Brandenburg, La Farge, Wis.; Bellevue Hospital Medical College, New York, 1885; aged 79; died, Nov. 17, 1941, in La Crosse.

Joseph Thompson Graham, Draper, Va.; Medical College of Virginia, Richmond, 1923; aged 45; died, Nov. 29, 1941, in the Pulaski (Va.) Hospital.

John D. Wilson, Bowie, Texas; Memphis (Tenn.) Hospital Medical College, 1892; aged 78; died, Nov. 17, 1941, in the Bowie Clinic Hospital.

John Curtis Black, Bradenton, Fla.; University of Arkansas School of Medicine, Little Rock, 1911; aged 54; died, Nov. 26, 1941, in Corning, Ark.

John H. McCain ● Arcola, Ill.; University of Louisville (Ky.) Medical Department, 1891; aged 72; died, Nov. 24, 1941, of cerebral hemorrhage.

Lilla B. Wood, Reed City, Mich.; Saginaw (Mich.) Valley Medical College, 1901; aged 74; died, Nov. 26, 1941, in the Reed City Hospital.

William G. Mullarky, Huntington, W. Va.; State University of Iowa College of Medicine, Iowa City, 1887; aged 83; died, Nov. 14, 1941.

Jack Sawyer Shipp, Anniston, Ala.; Louisiana State University School of Medicine, New Orleans, 1940; aged 35; died, Nov. 16, 1941.

Francis Conrad Vogt ● Brooklyn; Long Island College Hospital, Brooklyn, 1900; aged 62; died, Nov. 19, 1941, of heart disease.

William Henry Hodges, Finger, Tenn.; University of Nashville Medical Department, 1898; aged 72; died, Nov. 16, 1941.

Theophile Hubert Wilson, Buffalo; Trinity Medical College, Toronto, Ont., Canada, 1900; aged 65; died, Nov. 30, 1941.

Robert Lee Justice, Haines City, Fla.; Medical College of Alabama, Mobile, 1894; aged 70; died in November, 1941.

J. Mills Boal, San Diego, Calif.; New York Homeopathic Medical College, New York, 1884; aged 85; died, Oct. 3, 1941.

Backus M. McIntyre, Winside, Neb.; John A. Creighton Medical College, Omaha, 1905; aged 70; died, Nov. 8, 1941.

William Riley Tanner, Lubbock, Texas (licensed in Texas, under the Act of 1907); aged 89; died, Nov. 13, 1941.

Charles Barber Ballard, Marietta, Ohio; Miami Medical College, Cincinnati, 1889; aged 80; died, Nov. 7, 1941.

Cyrus S. Keagy, Mill Shoals, Ill.; Missouri Medical College, St. Louis, 1889; aged 78; died, Nov. 19, 1941.

William A. Wilson, Detroit; Detroit College of Medicine, 1895; aged 71; died, Nov. 15, 1941.

Deaths

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Francis LeRoy Phillips ● Assistant Surgeon, Lieutenant (junior grade) United States Navy, Mare Island, Calif.; University of Southern California School of Medicine, Los Angeles, 1936; entered the navy in August 1936; on the staff of the United States Naval Hospital; aged 32; died, Nov. 17, 1941, in the United States Naval Hospital, Brooklyn.

Theodore Henry Lemmers, Jersey City, N. J.; New York Homeopathic Medical College and Hospital, New York, 1896; fellow of the American College of Surgeons; member of the Medical Society of New Jersey; ophthalmologist and director of clinics, Christ Hospital; aged 71; died, Nov. 8, 1941, of carcinoma of the rectum.

William Henry Marsh, Solomons, Md.; University of Maryland School of Medicine, Baltimore, 1876; member of the Medical and Chirurgical Faculty of Maryland; formerly associated with the U. S. Public Health Service; aged 90; died, Nov. 12, 1941, in the United States Marine Hospital, Baltimore, of carcinoma of the lung.

Thomas Douglass ● Ozark, Ark.; Missouri Medical College, St. Louis, 1889; president of the Tenth Councilor District Medical Society and secretary of the Franklin County Medical Society; for many years chairman for the Red Cross in Franklin County; aged 73; died, Nov. 7, 1941, in a hospital at Fort Smith.

Albert Clinton Leach, Orange, Mass.; Dartmouth Medical School, Hanover, N. H., 1894; member of the Massachusetts Medical Society; member of the local board of health for many years; aged 70; died, Nov. 9, 1941, in the Farren Memorial Hospital, Montague City, of cerebral hemorrhage.

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Ernest Maxwell Vardon, Los Angeles; University of Michigan Department of Medicine and Surgery, Ann Arbor, 1898; member of the California Medical Association; on the staff of the Queen of Angels Hospital; aged 71; died, Oct. 8, 1941, of retroperitoneal hemorrhage.

Douglas Hursh Vastine, Catawissa, Pa.; University of Pennsylvania School of Medicine, Philadelphia, 1923; member of the Medical Society of the State of Pennsylvania; aged 42; died, Nov. 14, 1941, in the Bloomsburg (Pa.) Hospital, of pneumonia and meningitis.

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Jim Hilliard Camp ● Pecos, Texas; University of Texas School of Medicine, Galveston, 1927; at one time instructor in anatomy at his alma mater; medical director of a hospital bearing his name; aged 39; was accidentally shot and killed, Nov. 16, 1941.

David Henry Davison, New York; College of Physicians and Surgeons, medical department of Columbia College, New York, 1876; member of the Medical Society of the State of New York; aged 87; died, Nov. 22, 1941, in the Mount Sinai Hospital.

William August Kriesel, Little Rock, Ark.; Minneapolis College of Physicians and Surgeons, 1897; member of the Arkansas Medical Society; served during the World War; on the staff of the Missouri Pacific Hospital; aged 71; died, Oct. 30, 1941.

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Mavity J. Spencer, Indianapolis; Central College of Physicians and Surgeons, Indianapolis, 1896; member of the Indiana State Medical Association; past president of the city board of health; formerly police surgeon; aged 70; died, Nov. 27, 1941, of coronary embolism.

Harry Theodale Rosenthal, New York; University and Bellevue Hospital Medical College, New York, 1911; member of the Medical Society of the State of New York; on the staff of the Bronx Hospital; aged 54; died, Nov. 23, 1941, of coronary thrombosis.

Lloyd Henry Sarchet, Wellington, Kan.; State University of Iowa College of Homeopathic Medicine, Iowa City, 1898; member of the Kansas Medical Society; city and county health officer; aged 69; died, Nov. 14, 1941, in St. Luke's Hospital.

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Mace Anderson Losee, Livingstonville, N. Y.; Cornell University Medical College, New York, 1902; member of the

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Eugene Le Roy Kellum, Richmond, Va.; University of Pennsylvania School of Medicine, Philadelphia, 1924; on the staff of the Grace Hospital; aged 43; was killed, Nov. 15, 1941, in an automobile accident.

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Mitchell Lawrence Moran, St. Petersburg, Fla.; Georgetown University School of Medicine, Washington, D. C., 1935; member of the Florida Medical Association; aged 31; died, Nov. 22, 1941.

Charles Henry Wilson, New Brighton, Pa.; Western Pennsylvania Medical College, Pittsburgh, 1906; aged 69; died, Nov. 1, 1941, in the Beaver Valley Hospital of carcinoma of the stomach.

Harvy Nally, Cainsville, Mo.; University of Missouri School of Medicine, Columbia, 1876; member of the Missouri State Medical Association; aged 86; died, Nov. 3, 1941, of coronary thrombosis.

Eugene Leffler Gilmore, Tallapoosa, Ga.; Marion-Sims College of Medicine, St. Louis, 1892; member of the Medical Association of Georgia; aged 72; died, Nov. 16, 1941, of septicemia.

K. Arvid Enlind, New York; College of Physicians and Surgeons, Baltimore, 1893; served during the World War; aged 68; died, Nov. 17, 1941, in the Veterans Administration Facility.

Robert Graham Contrell, New York; University of Pennsylvania Department of Medicine, Philadelphia, 1896; aged 68; died, Nov. 26, 1941, in Englewood, N. J., of carcinoma of the prostate.

Arthur Horton Mann ● Silver City, N. M.; University of Pennsylvania School of Medicine, Philadelphia, 1919; aged 48; died, Nov. 2, 1941, of lobar pneumonia and pulmonary tuberculosis.

James Stanley Gardner, Winnipeg, Man., Canada; University of Manitoba Faculty of Medicine, Winnipeg, 1923; aged 44; died, Nov. 25, 1941, of herpes zoster and pulmonary tuberculosis.

Calvin H. Maust, Lecompton, Kan.; Central Medical College of St. Joseph, Mo., 1905; member of the Kansas Medical Society; aged 63; died, Nov. 4, 1941, in a hospital at Topeka.

David H. Thornton, Princeton, W. Va.; College of Physicians and Surgeons, Baltimore, 1893; aged 76; died, Nov. 19, 1941, in Roanoke, Va., of coronary sclerosis and hypertension.

Paul Alexander Johnstone ● Kansas City, Mo.; University Medical College of Kansas City, Mo., 1903; aged 75; died, Nov. 9, 1941, of coronary occlusion and pulmonary embolism.

Hartwell Blount Hyde, Nashville, Tenn.; Vanderbilt University School of Medicine, Nashville, 1878; aged 88; died, Nov. 12, 1941, in the Davidson County Hospital of senility.

John William Smart, Cottontown, Tenn.; University of Tennessee Medical Department, Nashville, 1886; aged 80; died, Nov. 16, 1941, in the Protestant Hospital at Nashville.

Joseph M. Hale, Dearborn, Mo.; Ensworth Medical College, St. Joseph, 1891; member of the Missouri State Medical Association; aged 77; died, Nov. 10, 1941, in Kansas City.

Lawrence Miles Gwinn ● Denver; University of Colorado School of Medicine, Denver, 1913; on the staff of the Presbyterian Hospital; aged 53; died, Nov. 30, 1941.

Richard Churchill Travis ● Indianapolis; University of Michigan Medical School, Ann Arbor, 1925; served during the World War; aged 42; hanged himself, Nov. 23, 1941.

John Dana Robbins, Mount Ida, Ark.; Memphis (Tenn.) Hospital Medical College, 1902; member of the Arkansas Medical Society; aged 68; died, Oct. 29, 1941.

Charles Henry Walter, Oakland, Calif.; Hahnemann Medical College and Hospital of Philadelphia, 1894; served during the World War; aged 74; died, Nov. 22, 1941.

Milton Elliott Cannon, Riceville, Tenn.; Lincoln Memorial University Medical Department, Knoxville, 1910; served during the World War; died, Nov. 28, 1041.

Charles J. Ross, Dover, Ark.; Memphis (Tenn.) Hospital Medical College, 1892; past president of the Pope County Medical Society; aged 74; died, Nov. 15, 1941.

George McGowan Stuart, Cupar, Sask., Canada; Manitoba Medical College, Winnipeg, Man., 1906; chairman of the public school board; aged 59; died, Oct. 28, 1941.

George Lewis Fuquay, Coats, N. C.; North Carolina Medical College, Charlotte, 1916; served during the World War; aged 49; died, Nov. 28, 1941.

Henry M. Kerzman, Detroit; Universität Bern Medizinische Fakultät, Switzerland, 1921; aged 53; died, Nov. 22, 1941, in the Harper Hospital.

Frederick David Brandenburg, La Farge, Wis.; Bellevue Hospital Medical College, New York, 1885; aged 79; died, Nov. 17, 1941, in La Crosse.

Joseph Thompson Graham, Draper, Va.; Medical College of Virginia, Richmond, 1923; aged 45; died, Nov. 29, 1941, in the Pulaski (Va.) Hospital.

John D. Wilson, Bowie, Texas; Memphis (Tenn.) Hospital Medical College, 1892; aged 78; died, Nov. 17, 1941, in the Bowie Clinic Hospital.

John Curtis Black, Bradenton, Fla.; University of Arkansas School of Medicine, Little Rock, 1911; aged 54; died, Nov. 26, 1941, in Corning, Ark.

John H. McCain ● Arcola, Ill.; University of Louisville (Ky.) Medical Department, 1891; aged 72; died, Nov. 24, 1941, of cerebral hemorrhage.

Lilla B. Wood, Reed City, Mich.; Saginaw (Mich.) Valley Medical College, 1901; aged 74; died, Nov. 26, 1941, in the Reed City Hospital.

William G. Mullarky, Huntington, W. Va.; State University of Iowa College of Medicine, Iowa City, 1887; aged 83; died, Nov. 14, 1941.

Jack Sawyer Shipp, Anniston, Ala.; Louisiana State University School of Medicine, New Orleans, 1940; aged 35; died, Nov. 16, 1941.

Francis Conrad Vogt ● Brooklyn; Long Island College Hospital, Brooklyn, 1900; aged 62; died, Nov. 19, 1941, of heart disease.

William Henry Hodges, Finger, Tenn.; University of Nashville Medical Department, 1898; aged 72; died, Nov. 16, 1941.

Theophile Hubert Wilson, Buffalo; Trinity Medical College, Toronto, Ont., Canada, 1900; aged 65; died, Nov. 30, 1941.

Robert Lee Justice, Haines City, Fla.; Medical College of Alabama, Mobile, 1894; aged 70; died in November, 1941.

J. Mills Boal, San Diego, Calif.; New York Homeopathic Medical College, New York, 1884; aged 85; died, Oct. 3, 1941.

Backus M. McIntyre, Winside, Neb.; John A. Creighton Medical College, Omaha, 1905; aged 70; died, Nov. 8, 1941.

William Riley Tanner, Lubbock, Texas (licensed in Texas, under the Act of 1907); aged 89; died, Nov. 13, 1941.

Charles Barber Ballard, Marietta, Ohio; Miami Medical College, Cincinnati, 1889; aged 80; died, Nov. 7, 1941.

Cyrus S. Keagy, Mill Shoals, Ill.; Missouri Medical College, St. Louis, 1889; aged 78; died, Nov. 19, 1941.

William A. Wilson, Detroit; Detroit College of Medicine, 1895; aged 71; died, Nov. 15, 1941.

Correspondence

PARALDEHYDE OR BENZYL ALCOHOL AS CAUSE OF FATALITY

To the Editor:—Since its introduction in obstetrics, paraldehyde has proved to be a valuable analgesic agent in labor. In addition to its excellent analgesic and amnesic properties, its usefulness has been greatly enhanced by the relaxation and sleep it affords the parturient patient between pains. Most physicians who have used this drug have been impressed with the wide margin of safety it enjoys over other commonly employed analgesics. In the hands of different obstetricians, and with varying technics of administration, it has been found to possess a high degree of safety for both mother and child. Hanson (*California & West. Med.* **47**:191 [Sept.] 1937) has reported a case in which an overdose (120 cc.) of paraldehyde was given rectally during labor, with complete recovery of the mother and the delivery of a normal infant in good condition. Yet reports of 4 cases have appeared in the literature incriminating paraldehyde, either directly or indirectly, as the cause of maternal death. Paraldehyde may be exonerated at once in the first case, for three rectal instillations of formaldehyde were given by mistake instead of the intended paraldehyde medication (Kane, H. F.; Roth, G. B., and Mandy, T. E.: *Delaware State M. J.* **10**:197 [Sept.] 1938).

Kotz, Roth and Ryon (THE JOURNAL, June 25, 1938, p. 2145) have attributed the death of their patient to an idiosyncrasy to paraldehyde. Although she became critically ill soon after receiving a single dose of 31 cc., death occurred only after cesarean section was performed. One may question, therefore, what the outcome might have been had a conservative course of treatment been followed.

Most recent is the report of Shoor (THE JOURNAL, Nov. 1, 1941, p. 1534). This author administered 12 cc. of paraldehyde and 6 cc. of benzyl alcohol to a primigravida aged 21 who had been in labor for about thirty-one hours. Three and one-half hours later she was found cyanotic and dyspneic and died following a generalized convulsion twenty-one and one-half hours after receiving the paraldehyde. The baby also died shortly after delivery, it too suffering generalized convulsions. Shoor, like Kotz, Roth and Ryon, attributed the maternal death to an idiosyncrasy to paraldehyde. It would appear unlikely that the fetus shared this "idiosyncrasy," but in view of both mother and child having undergone a similar mode of death a common cause of death seems reasonable. This, I believe, can be found in the benzyl alcohol. Used in conjunction with rectal instillations of paraldehyde, benzyl alcohol serves as a local surface anesthetic to the rectal mucosa. Kane and Roth (THE JOURNAL, Nov. 21, 1936, **107**, p. 1710) have recommended a constant dose of 1.5 cc. When one of their patients was given, by mistake, 115 cc. of this drug, death ensued. Benzyl alcohol, which acts as a protein precipitant in the body, has been found lethal for mice, kittens and guinea pigs in doses of from 1 to 2 cc. per kilogram of body weight. When toxic amounts of this drug were injected into animals, convulsions often resulted (Macht, D. I.: *J. Pharmacol. & Exper. Therap.* **11**:263 [April] 1918).

It is therefore submitted that Shoor's patient succumbed probably as a result of an excessive dose of benzyl alcohol, a substance of relatively high toxicity, rather than because of an idiosyncrasy to paraldehyde. Although reports of isolated cases exist in the literature, attributing fatal reactions to therapeutic doses of paraldehyde, there is no incontrovertible evidence that a single maternal death has ever resulted directly from the proper use of this valuable analgesic agent in obstetrics.

HAROLD SPEERT, M.D., St. Louis.

SUGGESTIONS FOR THE POSSIBLE CONTROL OF THE AMERICAN SUMMER ENCEPHALITIDES

To the Editor:—A recent editorial in THE JOURNAL (New Developments in Knowledge of Encephalitis, October 18, p. 136) reviews recent knowledge of certain "seasonal" encephalitides affecting man.

Since the outbreak in Paris, Ill., in 1932, epidemic summer encephalitis, epidemiologically not dissimilar to that occurring in the spring in Russia and to that of the late summer and autumn in Japan and Russia, has become increasingly important as a disease of man in the United States, Canada and Mexico. Approximately 3,000 cases were reported during the summer of 1941. At least three viruses may be responsible for the American outbreaks—that named for the St. Louis epidemic and those of the eastern and western types of equine encephalomyelitis. These three known American viruses are so closely related epidemiologically and clinically that the St. Louis infection should no longer be set apart from the so-called equine diseases either in name or in thought. For example, a large survey on serum neutralization made in the Yakima Valley, Washington, indicates that in this endemic area the western virus and the St. Louis virus infect, in approximately like proportions, horses and many other mammals and birds (Hammon, W. M.; Gray, J. A.; Evans, F. C.; Izumi, E. M., and Lundy, H. W.: Western Equine and St. Louis Encephalitis Antibodies in the Sera of Mammals and Birds from an Endemic Area, *Science* **94**:305 [Sept. 26] 1941). Also intracerebral inoculation of horses with any of these viruses will produce encephalomyelitis (Cox, H. R.; Philip, C. B., and Kilpatrick, J. W.: Susceptibility of Horses to St. Louis Encephalitis Virus, *Pub. Health Rep.* **56**:1391 [July 4] 1941). Furthermore, the same species of mosquitoes was found infected with both the St. Louis and the western equine viruses (Hammon, W. M.; Reeves, W. C.; Brookman, B.; Izumi, E. M., and Gjullin, C. M.: Isolation of the Viruses of Western Equine and St. Louis Encephalitis from Culex Tarsalis Mosquitoes, *Science* **94**:328 [Oct. 3] 1941). Thus these three may be considered as one group in our discussion of general control measures.

Before the formulation and enforcement of control measures are seriously undertaken there should be a reasonably complete and well founded knowledge of the means of transmission and other important epidemiologic data. I shall briefly review the facts to see whether the profession has arrived at such a stage.

Mosquitoes were suspected as vectors of the eastern and western viruses, first on the basis of epidemiologic and then on laboratory evidence; both viruses have been repeatedly transmitted by Aedes mosquitoes in the laboratory (Davis, W. A.: A Study of Birds and Mosquitoes as Hosts for the Virus of Eastern Encephalomyelitis, *Am. J. Hyg.* **32**:45, sec. C [Sept.] 1940). The St. Louis virus, at the time it was first recognized, was considered by one group of investigators to be spread by the respiratory tract (Leake, J. P.; Musson, E. K., and Chope, H. D.: Epidemiology of Epidemic Encephalitis, St. Louis Type, THE JOURNAL, Sept. 8, 1934, p. 728) and by others as mosquito borne (Casey, A. E., and Broun, G. O.: Epidemiology of St. Louis Encephalitis, *Science* **88**:450 [Nov. 11] 1938. (Lumsden, L. L.: Unpublished official report). Japanese workers reported laboratory transmission with Culex pipiens (Mitamura, T.; Yamada, S.; Hazato, H.; Mori, K.; Hosoi, T.; Kitaoka, M.; Watanabe, S.; Okubo, K., and Temjin, S.: Ueber den Infektionsmodus der epidemischen Enzephalitis: Experimentelle Untersuchungen über ihre Ansteckung durch Mücken, *Tr. Jap. Path. Soc.* **27**:573, 1937), but in the United States this transmission has not been confirmed (Fulton, J. D.; Greutter, J. E.; Muether, R. O.; Hanss, E. B., and Broun, G. O.: Observations Concerning Culex Pipiens as a Possible Carrier of St. Louis Encephalitis, *Proc. Soc. Exper. Biol. & Med.*

66:255 [May] 1940). During the summer of 1941 in the Yakima Valley both the St. Louis and the western viruses were repeatedly isolated from Culex tarsalis (Hammon, W. M.; Reeves, W. C.; Brookman, B.; Izumi, E. M., and Gjullin, C. M., to be published). Thus insect transmission of all three types seems to be rather well established, though the final evidence of demonstrating the ability to transmit for the particular mosquito found naturally infected is still lacking.

The finding of western equine and of St. Louis virus in nasal washings of experimental horses by Records and Vawter (Equine Encephalomyelitis Cross Immunity in Horses Between Western and Eastern Strains of Virus, Supplemental Report, *J. Am. Vet. M. A.* **39**:773, 1935) and by Cox, Philip and Kilpatrick respectively and the ease of intranasal infection in the laboratory especially of the latter disease (Vawter, L. R., and Records E.: Respiratory Infection in Equine Encephalomyelitis, *Science* **78**:41 [July 14] 1933) makes it unwise to deny the possibility of this route of infection altogether. The importance of finding virus in the nasal washings of 1 horse from an experimentally induced St. Louis infection (Cox, Philip and Kilpatrick) should not be exaggerated, for in our experiments the virus was readily found in the blood stream in 2 horses during inapparent infections and could not be detected in the nasal washings (Hammon, W. M.; Carle, B. N.; Izumi, E. M., and Britton, J. W.: St. Louis Encephalitis in the Horse, to be published). Thus accumulated epidemiologic and laboratory evidence points strongly to the greater importance of transmission by insects.

Conclusive evidence now indicates that these viruses are widespread in smaller animals, the eastern type having been isolated from three species of birds and the western type from a prairie chicken (Cox, H. R.; Jellison, W. L., and Hughes, L. E.: Isolation of Western Equine Encephalomyelitis Virus from a Naturally Infected Prairie Chicken, *Pub. Health Rep.* **56**:1905 [Sept. 26] 1941). Preliminary tabulation of results of neutralization tests on about 300 serums from mammals and birds in the Yakima Valley, Washington (Hammon, Gray, Evans, Izumi and Lundy), now controlled by a series of about 150 bloods from non-encepnalitis areas, indicates that approximately 50 per cent of all domestic animals from this endemic area as well as about 12 per cent of the sampled members of wild species are infected at some time with both the St. Louis and the western virus (Hammon, Gray, Evans, Izumi and Lundy). This widespread infection occurs in most instances without any apparent epizootic except in horses.

Reports of the disease in horses and man, frequent instances of the isolation of the viruses and serologic tests give data of a useful nature regarding the geographic distribution of the infections and suggest that new areas are being involved each year and that the disease is reappearing in areas apparently free from it for long periods of time. The western virus has now been reported from every state west of the Mississippi River and from about three fourths of all counties within these states, as well as in numerous areas east of this line. Mass vaccination of horses, though partly responsible for fewer total cases in this species since 1938, has not checked the apparent spread. Since knowledge of the St. Louis virus has until now been limited to its recognition in human beings, less is known of its distribution, but such evidence as is available suggests that it is present in many areas of both the East and the West. The eastern infection appeared for a time to be limited to the area east of the Appalachian Mountains but in 1940 was found in Alabama and in 1941 in several parts of Texas. In the Rio Grande Valley during the summer of 1941 I obtained serologic evidence of both eastern and western virus infections in nonfatal cases in man and of all three types in horses.

With this knowledge of the means of transmission and of the apparent spread and distribution, it would seem that physicians are now ready to face the important problem of what they can and will do about it. The large irrigated areas of certain Western states are known to offer ideal conditions for annual epidemics. The eastern type virus is by far the most dangerous to horse and man and seems to be rapidly moving westward and may appear next year or the year after in the great San Joaquin and Sacramento valleys of California and in other similar areas, where it can be expected to become endemic. Can it be prevented from doing so? Can it be prevented from entering the Dakotas and the great Northwest, including portions of Canada? Can it be prevented from recurring in Massachusetts and other eastern areas? In areas of the Western and Central states in which the western virus is now firmly established, can the disease be controlled? Can the three viruses now present in Texas on the Gulf Coast and the lower Rio Grande Valley, first observed in man in 1941, be prevented from causing epidemics in the large concentrations of Army and Navy personnel located in these areas?

I shall consider the more probable means of spread from one area to another: 1. The shipment of domestic animals just after natural infection could result in mosquito infection at any point at which these animals might be delivered from one to six days after shipment, given the vector and other suitable environmental conditions. This is evident from the fact that inoculation of birds and small mammals in the laboratory by the subcutaneous or intracutaneous route (similar to insect inoculation) or by intracerebral inoculation results in the virus circulating in the blood during some period from twelve to ninety-six hours after inoculation (Ten Broeck, Carl: Birds as Possible Carriers of the Virus of Equine Encephalomyelitis, *Arch. Path.* **25**:759 [May] 1938. Howitt, B. F.: Comparative Susceptibility of Wild and Domestic Birds and Animals to the Western Virus of Equine Encephalomyelitis (Br. Strain) in California, *J. Infect. Dis.* **67**:177 [Nov.-Dec.] 1941. Davis). In horses it may appear as late as six days after inoculation (Records and Vawter). 2. Given an infected mosquito, the possibility of its transportation by airplane, train, automobile or boat must be considered. 3. Migratory birds in an area such as the lower Rio Grande Valley, in which the infection occurs in horses as early as March, might become infected just before beginning flight northward and serve to infect mosquitoes at certain places where the birds temporarily rest.

To counteract these three possible methods of spread, the following might be considered: 1. A quarantine period for domestic animals shipped during the summer from known infected areas. This should be of about four days for small and six days for large animals and would have to be in mosquito protected quarters. There is no evidence at present to indicate a prolonged carrier state in any except an arthropod host, so there is no justification for a prolonged quarantine period. 2. Mosquito inspection and spraying are now practiced in yellow fever and malaria areas for air, water and land vehicles and could be applied to encephalitis areas. California already has entomologic inspection stations at important points of entry to the state, and these agricultural protective measures might be extended to protect man and horses from the highly fatal eastern encephalomyelitis virus. 3. Migratory birds would still jeopardize the situation even if these other measures were put into effective practice. Outside of extermination of these, a thought that can be dismissed entirely, intense local control measures in infected areas, to be considered later, would reduce the likelihood of birds becoming infected in southern endemic regions.

Undoubtedly a carefully planned and well correlated program which envisions the problem as a national or international one, rather than one of states or counties alone, could change the natural course of events.

Not less important, and not unrelated, is the question of local control where one or more of these viruses is now established.

When one considers the vast extent of the reservoir and the difficulties involved in complete elimination of mosquitoes in irrigated areas, it is obvious that the problem is somewhat similar to that of sylvatic plague—to learn how best to live with it rather than how to eradicate it. The following possibilities are pointed out:

1. As soon as the particular arthropod vector or vectors of the region are incriminated, and immediate steps should be taken to do so, their control should be considered. In most instances at least partial control will be practicable. If Culex tarsalis is the vector, however, owing to its ability to breed under such varied conditions, its effective control in certain areas will be entirely impracticable. Careful screening of homes and avoidance of mosquito infected areas during the hours when they customarily bite will offer partial protection. Those persons who, because of occupation, cannot remain indoors at these hours should wear protective clothing and use repellants, of which reasonably satisfactory types are now available.

2. Vaccination of man is being practiced on a small scale at present and deserves careful evaluation. Vaccination of horses for both the eastern and the western virus has proved highly effective. It is practicable for these animals because the morbidity rate without vaccination is usually about 10 per cent and the fatality rates are from 30 to 90 per cent. If an occasional horse dies or suffers a severe reaction from vaccination, it is relatively unimportant. In man, however, morbidity rates in the reported outbreaks (reports incomplete for 1941) have ranged from less than 1 to a maximum of about 150 per hundred thousand for a county or up to 500 per hundred thousand for a smaller political subdivision. Mortality has varied from 3 to 30 per cent in the western and St. Louis outbreaks. Let us consider how many persons would require vaccination for each life saved in what is considered a severe and unusual outbreak. Had the outbreak in St. Louis County and City in 1933 (morbidity rate 100 per hundred thousand and case fatality rate 20 per cent) been predicted and a perfect vaccine been available, for each life saved 5,000 persons would have been vaccinated. In an area such as the Yakima Valley, where annual vaccination of the population might be considered, because the disease appears annually, over the last three years for each life saved 13,900 vaccinations would have been performed. Until the outbreak involving the Dakotas in 1941, the morbidity rates in this area have been higher than anywhere else in the United States. Since at least two viruses are present in the area, double vaccination would probably have been necessary. In the most heavily infected counties of the San Joaquin Valley of California, much greater numbers would require vaccination for each life one would expect to save. Moreover, in this area the migratory population most needing vaccination would present an extremely difficult administrative problem. Most epidemics, however, are not predictable and, when recognized and the virus or viruses responsible identified in the laboratory, the time for vaccination to be effective would probably have passed. Since man is an unimportant reservoir, vaccination of a part of a population would not appreciably decrease the chance of infection for the unvaccinated group, as it does in smallpox. Some untoward reactions can be expected in every few thousand vaccinations, including the possibility of death, as is the case with any vaccine now used on human beings and with these specific vaccines when used on horses. Thus it would seem premature to recommend general vaccination of man in epidemic or endemic areas and not advisable, unless morbidity rates increase greatly above those so far reported, to compare with those rates encountered in certain communities in Russia, where vaccination is practiced for their spring, tick-borne encephalitis. There, in one community in which there were unvaccinated controls, it appears that 1 life was saved for every group of 267 persons vaccinated. Selected groups of heavily exposed persons, probably on an occupational basis, such as certain laboratory workers or agricultural workers, might well be immunized. This is now being practiced for the eastern and western viruses with reasonably good evidence that it is effective (Beard, D.; Finkelstein, H., and Beard, J. W.: Repeated Vaccination of Man Against the Virus of Equine Encephalomyelitis, *J. Immunol.* **40**:492 [April] 1941). However, vaccination must be repeated yearly. At present no vaccine is available for the St. Louis infection.

3. Zoning restrictions to eliminate domestic stock and fowls from cities, suburban areas and small towns would undoubtedly be an effective measure in reducing human morbidity rates and deserves serious and immediate consideration. The distribution of the human disease and the preponderance of infection in domestic animals present strong evidence pointing to close association of man with a concentrated population of domestic animals as an essential to a high morbidity rate. In large cities, where zoning regulations are now enforced, despite the large human population few cases occur, and these can usually be traced to exposure elsewhere (Hammon, W. M., and Howitt, B. F.: Epidemiological Aspects of Encephalitis in the Yakima Valley, Washington: Mixed St. Louis and Western Equine Types, *Am. J. Hyg.*, to be published). Such control measures, although they will result in certain hardships on persons engaged in raising poultry or keeping stock of any kind in residential districts, are practicable and would undoubtedly be highly effective.

4. Animals that would be difficult to eliminate, such as dogs, and all animals in areas in which owners of small tracts live under conditions closely simulating rural towns could probably be prevented from serving as reservoirs of virus by vaccination. Technics and vaccines would have to be developed, but this could be readily accomplished. In cases in which placental or colostrum transmitted antibodies cannot play a role, the young of each species—especially fowl—would require vaccination soon after birth and thus make vaccination expensive and annoying to the farmer.

5. Robins, pheasants, quail and a few other species of wild life have high infection rates and may well serve to infect mosquitoes in areas inhabited by man. The wild life group and sportsmen should gradually familiarize themselves with the facts and realize that something should be done about it. Probably, however, by controlling the domestic reservoirs the infection rate in mosquitoes and, in turn, in these wild birds will be greatly reduced. It would seem wise, however, to discontinue heavy planting of pheasants and perhaps certain other game in endemic areas. In the Yakima Valley, fourteen thousand pheasants a year are being released, and those caught in this area appear to have an infection rate approaching 50 per cent.

The control measures outlined, both for preventing the entry of a new virus and for the control of those already present, are offered at this time only as suggestions. Some of the evidence on which they are based may yet be shown to be in error or only part of the truth. Some are more or less impracticable from the administrative standpoint. Some are warranted in one area and not in another. It has been my purpose to impress those responsible for the prevention and control of disease, and those who might serve to influence them, with the immediate and pressing importance of the situation and to outline briefly the measures that could be employed, based on our present knowledge. An attempt has been made to evaluate some of these procedures. The situation has grave potentialities, threatening both civilian and military horses and personnel. It is increasing yearly in importance, and prompt, intelligent action can undoubtedly change the course of events.

WILLIAM MCDOWELL HAMMON, M.D., DR.P.H.,
George Williams Hooper Foundation,
University of California, San Francisco.

Council on Medical Education and Hospitals

CONTINUATION COURSES FOR PRACTICING PHYSICIANS

In accordance with the plan of the Council on Medical Education and Hospitals, advance information concerning continuation courses for practicing physicians available in the various centers is published quarterly. The following list of courses is presented for the period Jan. 1 to April 30, 1942. It is hoped that this material will be of value to the practicing physician who may be planning to take postgraduate work but who does not have at hand a ready means of knowing when and where the subjects in which he is interested will be taught. It is urged that those who contemplate enrolling in any of these courses communicate as early as possible with the executive officer named in the following list as the size of classes is in many cases necessarily limited.

WILLIAM D. CUTTER, M.D.,
Secretary, Council on Medical
Education and Hospitals.

Continuation Courses for Practicing Physicians—January 1 to April 30, 1942

Institution	Courses Scheduled to Begin	Length and Content of Course	Number of Students Accepted for Each Course	Registration Fee and/or Tuition	For Detailed Information, Write to
ALLERGY					
Washington University School of Medicine and Barnes Hospital, St. Louis	April 6	2 weeks	6-8 [1]	$40	Executive Secretary, American College of Physicians, 4200 Pine St., Philadelphia
Faculty of Medicine of Columbia University	April 13	3 weeks	4-8	$150	Dean of the School of Medicine, Columbia University, 630 West 168th St., New York City
Roosevelt Hospital, New York	February 2	2 weeks	6-8 [1]	$40	Executive Secretary, American College of Physicians, 4200 Pine St., Philadelphia
Graduate School of Medicine, University of Pennsylvania	Arranged on application	4 weeks, 40 hours	.....	$150	Dean, Graduate School of Medicine, University of Pennsylvania, Philadelphia
ANATOMY					
College of Medical Evangelists	Arranged on application	5 to 10 hours; elective subcourses	.....	$5 to $15	Chairman, Committee on Postgraduate Education, 312 N. Boyle Ave., Los Angeles
University of Illinois College of Medicine	Arranged on application	Variable	4 or more	$75 a semester plus cost of materials	Assistant to the Dean, University of Illinois College of Medicine, 1853 W. Polk St., Chicago
New York Medical College	Arranged on application	60 to 100 hours (surgical anatomy)	1 or more	$150 to $250	Dean, New York Medical College, 5th Ave. at 105th St., New York City
ANESTHESIA					
Harvard Medical School, Courses for Graduates	Arranged on application	1 month	3	$30 [2]	Assistant Dean, Harvard Medical School, 25 Shattuck St., Boston
Faculty of Medicine of Columbia University	Arranged on application [3]	2 or 3 weeks	.....	$100 to $150	Dean of the School of Medicine, Columbia University, 630 W. 168th St., New York City
New York Polyclinic Medical School and Hospital	Arranged on application	6 week and 3 month courses	.....	$150 to $300	Medical Executive Officer, New York Polyclinic Medical School, 345 W. 50th St., New York City
New York University College of Medicine	Spring	3 weeks (inhalation)	5	$150	Assistant Dean, New York University College of Medicine, 477 First Ave., New York City
		3 weeks, mornings or full time (regional)	.....	$200	
ARTHRITIS					
Tufts Medical School, Postgraduate Division	March 2	6 days	.....	$25 [2]	Chairman, Postgraduate Division, Tufts Medical School, 20 Bennet St., Boston
The Mayo Foundation, University of Minnesota and The Mayo Clinic, Rochester	April 13	1 week	20-35 [1]	$20	Executive Secretary, American College of Physicians, 4200 Pine St., Philadelphia
Faculty of Medicine of Columbia University	March 2	5 days	4 or more	$35	Dean of the School of Medicine, Columbia University, 630 W. 168th St., New York City
AVIATION MEDICINE					
George Washington University School of Medicine	February 5	3 days	.....	$35	Professor of Ophthalmology, George Washington University School of Medicine, 1335 H St., N.W., Washington, D. C.
BACTERIOLOGY					
Faculty of Medicine of Columbia University	January; February	1 month, mornings; part time courses	3-8	$50	Dean of the School of Medicine, Columbia University, 630 W. 168th St., New York City
BRONCHO-ESOPHAGOLOGY					
Harvard Medical School, Courses for Graduates	Arranged on application	2 weeks (bronchoscopy)	.....	$150 [2]	Assistant Dean, Harvard Medical School, 25 Shattuck St., Boston
Faculty of Medicine of Columbia University	Arranged on application	3 weeks	.....	$250	Dean of the School of Medicine, Columbia University, 630 W. 168th St., New York City
Temple University School of Medicine	March 16	2 weeks	.....	$250	Dean, Temple University School of Medicine, 3400 N. Broad St., Philadelphia
Graduate School of Medicine, University of Pennsylvania	January	2 weeks, 85 hours	.....	$250	Dean, Graduate School of Medicine, University of Pennsylvania, 36th and Pine Sts., Philadelphia
CARDIOLOGY					
College of Medical Evangelists	January 4	30 hours arranged	5-10	$50	Chairman, Committee on Postgraduate Education, 312 N. Boyle Ave., Los Angeles
Massachusetts General Hospital and House of the Good Samaritan, Boston	February 2	2 weeks	20-30 [1]	$40	Executive Secretary, American College of Physicians, 4200 Pine St., Philadelphia
Jewish Hospital, Brooklyn (Joint Committee on Post-Graduate Education)	January 13	5 weeks, 3 mornings a week	6	$20	Registrar, Joint Committee on Post-Graduate Education, 1313 Bedford Ave., Brooklyn
	April 7	5 weeks, 3 mornings a week (includes applied electrocardiography)	12	$20	

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Continuation Courses for Practicing Physicians—January 1 to April 30, 1942—Continued

Institution	Courses Scheduled to Begin	Length and Content of Course	Number of Students Accepted for Each Course	Registration Fee and/or Tuition	For Detailed Information, Write to
CARDIOLOGY—Continued					
Faculty of Medicine of Columbia University	April 6	4 weeks	10 or more	$100	Dean of the School of Medicine, Columbia University, 630 W. 168th St., New York City
New York Medical College.....	Arranged on application	8 weeks, twice a week (includes electrocardiography)	.....	$100	Dean, New York Medical College, 5th Ave. at 105th St., New York City
DERMATOLOGY					
Harvard Medical School, Courses for Graduates	February; April	1 month, mornings	Limited	$40 [2]	Assistant Dean, Harvard Medical School, 25 Shattuck St., Boston
	Arranged on application	2 months, twice a week (clinical mycology)	6	$50 [2]	
Tufts Medical School, Postgraduate Division	January 19	6 days	6 or more	$25 [2]	Chairman, Postgraduate Division, Tufts Medical School, 30 Bennet St., Boston
Faculty of Medicine of Columbia University	Arranged on application	6 weeks or 3 months, 3 afternoons a week	3	$40; $75	Dean of the School of Medicine, Columbia University, 630 W. 168th St., New York City
	Arranged on application [3]	3 months or more, 3 sessions a week (histology)	12	$75 to $175	
		6 weeks or 3 months, 3 sessions a week (surgical)	2	$40; $75	
		6 weeks or 3 months, 3 sessions a week (mycology)	.....	$40; $75	
DIABETES					
Tufts Medical School, Postgraduate Division	January 19	6 days	.....	$25 [2]	Chairman, Postgraduate Division, Tufts Medical School, 30 Bennet St., Boston
Faculty of Medicine of Columbia University	March 9	5 days	4 or more	$35	Dean of the School of Medicine, Columbia University, 630 W. 168th St., New York City
Graduate School of Medicine, University of Pennsylvania	Arranged on application	2 to 4 weeks, 75 hours	.....	$150	Dean, Graduate School of Medicine, University of Pennsylvania, Philadelphia
ELECTROCARDIOGRAPHY					
Tufts Medical School, Postgraduate Division	January 26	3 days (advanced course)	.....	$20 [2]	Chairman, Postgraduate Division, Tufts Medical School, 30 Bennet St., Boston
Faculty of Medicine of Columbia University	April 7	4 weeks, twice a week (fundamentals)	4 or more	$50	Dean of the School of Medicine, Columbia University, 630 W. 168th St., New York City
ENDOCRINOLOGY					
Jewish Hospital, Brooklyn (Joint Committee on Post-Graduate Education)	April	5 weeks, twice a week	4	$20	Registrar, Joint Committee on Post-Graduate Education, 1313 Bedford Ave., Brooklyn
Faculty of Medicine of Columbia University	February 2	5 weeks, 3 afternoons a week (diseases of metabolism)	6 or more	$40	Dean of the School of Medicine, Columbia University, 630 W. 168th St., New York City
	March 16	2 weeks	6 or more	$50	
New York Medical College.....	Arranged on application	4 weeks, 36 hours	.....	$100	Dean, New York Medical College, 5th Ave. at 105th St., New York City
FORENSIC MEDICINE					
New York University College of Medicine	Arranged on application	1 month or more	.....	$25 to $100	Assistant Dean, New York University College of Medicine, 477 First Ave., New York City
GASTROENTEROLOGY					
University of Chicago, The School of Medicine, Billings Hospital	April 6	2 weeks	50-75 [4]	$40	Executive Secretary, American College of Physicians, 4200 Pine St., Philadelphia
	Arranged on application	2 week and 3 month courses in gastroscopy	3; 1	$100 to $150	Dean of Students, University of Chicago, The School of Medicine, Chicago
Tufts Medical School, Postgraduate Division	February 9	6 days	.....	$25 [2]	Chairman, Postgraduate Division, Tufts Medical School, 30 Bennet St., Boston
Greenpoint Hospital, Brooklyn (Joint Committee on Postgraduate Education)	February 2	6 weeks, twice a week	4	$10	Registrar, Joint Committee on Post-Graduate Education, 1313 Bedford Ave., Brooklyn
Jewish Hospital, Brooklyn (Joint Committee on Postgraduate Education)	April 7	4 weeks, twice a week	8	$10	
Faculty of Medicine of Columbia University	Arranged on application [3]	2 months, 3 afternoons a week (gastroscopy)	1	$200	Dean of the School of Medicine, Columbia University, 630 W. 168th St., New York City
	April 6	4 weeks	10 or more	$100	
New York Medical College.....	Arranged on application	10 sessions (gastroscopy)	.....	$100	Dean, New York Medical College, 5th Ave. at 105th St., New York City
New York Polyclinic Medical School and Hospital	January 2; April 1	6 weeks	.....	$75 to $100	Medical Executive Officer, New York Polyclinic Medical School, 335 W. 50th St., New York City
Hahnemann Medical College and Hospital	Arranged on application	1 month or more	.....	Varies	Head of Department of Gastroenterology, Hahnemann Medical College, Philadelphia
Graduate School of Medicine, University of Pennsylvania	Arranged on application [3]	16 weeks, 500 hours	.....	$400	Dean, Graduate School of Medicine, University of Pennsylvania, Philadelphia
Graduate Hospital, University of Pennsylvania, Philadelphia	February 2	6 days	20-48 [4]	$20	Executive Secretary, American College of Physicians, 4200 Pine St., Philadelphia
GYNECOLOGY—See also under Obstetrics					
Faculty of Medicine of Columbia University	January	Various courses throughout year	Limited	$40 to $300	Dean of the School of Medicine, Columbia University, 630 W. 168th St., New York City
New York Polyclinic Medical School	Arranged on application	6 weeks	.....	$75	Medical Executive Officer, New York Polyclinic Medical School, 335 W. 50th St., New York City
INDUSTRIAL MEDICINE AND SURGERY					
Faculty of Medicine of Columbia University	February 2	2 weeks	.....	$60	Dean of the School of Medicine, Columbia University, 630 W. 168th St., New York City

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Institution	Courses Scheduled to Begin	Length and Content of Course	Number of Students Accepted for Each Course	Registration Fee and/or Tuition	For Detailed Information, Write to
LARYNGOLOGY—See also Otolaryngology					
Harvard Medical School, Courses for Graduates	Arranged on application	5 exercises (resection of nasal septum)	Limited	$75 [2]	Assistant Dean, Harvard Medical School, 25 Shattuck St., Boston
	February 16	6 weeks (anatomy)	Limited	$250 [2]	
Graduate School of Medicine, University of Pennsylvania	Arranged on application	10 days, 20 hours (operations on cadaver)	.....	$150	Dean, Graduate School of Medicine, University of Pennsylvania, Philadelphia
	January	2 weeks (laryngeal surgery)	.....	$250	
MEDICINE—General Medical and Surgical Subjects					
Tuskegee Institute, Ala.	April	1 week (clinics for Negro physicians)	.....	$5	Medical Director, John A. Andrew Memorial Hospital, Tuskegee Institute, Ala.
University of California Medical School and Stanford University School of Medicine	February 2	2 weeks	20 or more [1]	$40	Executive Secretary, American College of Physicians, 4200 Pine St., Philadelphia
Midwinter Postgraduate Clinics, Denver, Colorado	February 19	3 days	.....	$5	Executive Secretary, Colorado State Medical Society, 537 Republic Building, Denver
University of Kansas School of Medicine	March 30	4 days	.....	No	Chairman, Postgraduate Courses, University of Kansas School of Medicine, 39th St. and Rainbow Blvd., Kansas City, Kan.
New Orleans Graduate Medical Assembly	March 2	5 days	.....	$10	Secretary, New Orleans Graduate Medical Assembly, 1430 Tulane Ave., New Orleans
St. Louis Clinics.....	March	4 days	.....	$10	St. Louis Clinics, 3839 Lindell Blvd., St. Louis
New York Polyclinic Medical School and Hospital	Arranged on application	6 weeks	.....	$100	Medical Executive Officer, New York Polyclinic Medical School, 345 W. 50th St., New York City
Eighth Post-Collegiate Assembly, Ohio State University	March 5	3 days	.....	$2 to $5	Dean, College of Medicine, Ohio State University, Columbus
University of Oregon Medical School, Alumni Association	March 22	7 days	.....	Not available	Dean, University of Oregon Medical School, 3181 S.W. Marquam Hill Road, Portland
Seventh Annual Postgraduate Institute, Philadelphia County Medical Society	April 13	5 days	.....	$5	Director, Seventh Annual Postgraduate Institute, 301 S. 21st St., Philadelphia
Fourteenth Annual Spring Clinical Conference, Dallas Southern Clinical Society	March 23	4 days	.....	Not available	Secretary, Dallas Southern Clinical Society, 1123 Medical Arts, Bldg., Dallas
Sixth Annual Post Graduate Assembly for Negro Physicians, Prairie View, Texas	March 2	4 days	.....	No	Executive Secretary, Texas Tuberculosis Association, 700 Brazos, Austin
University of Wisconsin Medical School	April 18	1 week	.....	$10	Dean, University of Wisconsin Medical School, Madison
MEDICINE, INTERNAL					
University of California Medical School	January 5	3 days (drug therapy)	.....	$20	Director of Refresher Courses, c/o Dean's Office, University of California Medical School, San Francisco
Tulane University of Louisiana Department of Graduate Medicine	January 5; February 16	6 weeks (review course)	.....	$150	Director of Graduate Medicine, Tulane University of Louisiana, 1430 Tulane Ave., New Orleans
West Baltimore General Hospital...	1st of each month	3 months, 6 hours a week	.....	$30 to $125	Director, Postgraduate Training, West Baltimore General Hospital, Rayner and Dukeland Aves., Baltimore
University of Maryland School of Medicine, Johns Hopkins University School of Medicine	February 2	2 weeks (chemotherapy, hematology, nutrition, endocrinology)	20-50 [1]	$40	Executive Secretary, American College of Physicians, 4200 Pine St., Philadelphia
Harvard Medical School, Courses for Graduates	April 1	1 month or more	.....	$150 a month [2]	Assistant Dean, Harvard Medical School, 25 Shattuck St., Boston
	Monthly	Varies (endoscopy)	2	Varies	
University of Minnesota Medical School	April 6	2 weeks	30-50 [1]	$40	Executive Secretary, American College of Physicians, 4200 Pine St., Philadelphia
Bellevue Hospital (New York University College of Medicine)	1st of each month	1 month, mornings	6-12	$50	Director, Fourth Medical Division, Bellevue Hospital, 26th St. and East River, New York City
Faculty of Medicine of Columbia University	February 16	5 days (chemotherapy and vitamins)	4 or more	$35	Dean of the School of Medicine, Columbia University, 630 W. 168th St., New York City
	January 5; April 6	1 or 2 months, daily	4-10	$125 or $200	
	January 19	2 weeks (diagnostic procedures)	4-10	$50	
New York Medical College.....	Arranged on application	8 weeks, twice a week (physical diagnosis and hematology)	.....	$100	Dean, New York Medical College, 5th Ave. at 105th St., New York City
		10 hours (peritoneoscopy)	.....	$75	
NEUROLOGY AND PSYCHIATRY					
Colorado Psychopathic Hospital (Colorado State Medical Society)	February 16	3 days	20	$10	Executive Secretary, Colorado State Medical Society, 537 Republic Building, Denver
University of Illinois College of Medicine	Arranged on application	Seminars in psychiatry	Limited	Not available	Assistant to the Dean, University of Illinois College of Medicine, 1853 W. Polk St., Chicago
Harvard Medical School, Courses for Graduates	April 14	6 weeks, 3 mornings a week (neuropathology)	Limited	Arranged	Assistant Dean, Harvard Medical School, 25 Shattuck St., Boston
Faculty of Medicine of Columbia University	April 6	1 month	4-6	$100	Dean of the School of Medicine, Columbia University, 630 W. 168th St., New York City
	Arranged on application	1 month or more, afternoons (clinical neurology)	6	$30 a month	

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NEUROLOGY AND PSYCHIATRY—Continued					
Graduate School of Medicine, University of Pennsylvania	Arranged on application [4]	10 weeks, 250 hours	.....	$100	Dean, Graduate School of Medicine, University of Pennsylvania, 36th and Pine Sts., Philadelphia
	Arranged on application	8 weeks, 240 hours	.....	$100	
OBSTETRICS					
The Chicago Maternity Center.....	January	4 months	.....	$10	Medical Director, The Chicago Maternity Center, 1336 N. Newberry Ave., Chicago
University of Chicago, The School of Medicine	January 12; February 9; March 9; April 13	4 weeks	7	$25	Postgraduate Course, Department of Obstetrics and Gynecology, 5841 Drexel Ave., Chicago
University of Illinois College of Medicine	Every other week	2 weeks (includes pediatrics)	4	$10 [9]	Assistant to the Dean, University of Illinois College of Medicine, 1853 W. Polk St., Chicago
	Arranged on application	Obstetrics and gynecology in preparation for board examinations	Limited	Arranged	
Indiana University Medical Center	January 12; April 13	2 weeks	6	$10 [9]	Department of Postgraduate Education, Indiana University Medical Center, Indianapolis
State University of Iowa College of Medicine	Every other week	6 days	4	No	Associate in Obstetrics and Gynecology, University Hospitals, Iowa City, Ia.
Louisiana State University School of Medicine	Quarterly	2 weeks	.....	$75 [9]	Supervisor, Maternal and Child Health Services, Department of Health, Civil Courts Building, New Orleans
Tulane University of Louisiana Department of Graduate Medicine	February 18	6 weeks (review course, includes gynecology)	.....	$150	Director, Department of Graduate Medicine, Tulane University of Louisiana, 1430 Tulane Ave., New Orleans
Harvard Medical School, Courses for Graduates	Monthly	1 month or more	6	$125 [9]	Assistant Dean, Harvard Medical School, 25 Shattuck St., Boston
University of Michigan Medical School	Every other week	2 weeks	4	No	Director, Bureau of Maternal and Child Health, Michigan Department of Health, Lansing
University of Nebraska School of Medicine	Every other week	2 weeks (includes gynecology and pediatrics)	.....	$10	Director of Maternal and Child Health, State Department of Health, Lincoln
Margaret Hague Maternity Hospital, Jersey City (Faculty of Medicine, Columbia University)	1st of each month	1 to 3 months	24	$100 to $250	Medical Director, Margaret Hague Maternity Hospital, 88 Clifton Pl., Jersey City, N. J.
New York Polyclinic Medical School and Hospital	April	2 months	.....	$250	Medical Executive Officer, New York Polyclinic Medical School, 345 W. 50th St., New York City
Duke Hospital, Durham.....	Weekly	5 days (includes pediatrics)	.....	No [9]	Assistant State Health Officer, State Board of Health, Raleigh, N. C.
University of Oklahoma School of Medicine	Arranged on application	15 days	6	$25	Dr. Edward N. Smith, Associate Professor of Obstetrics, 800 N.E. 13th St., Oklahoma City
University of Oregon Medical School	January 19	5 days (includes gynecology)	.....	Not available	Dean, University of Oregon Medical School, 3181 S.W. Marquam Hill Road, Portland
Medical College of the State of South Carolina	Every other week	2 weeks (includes pediatrics)	4	$50 [9]	Dean, Medical College of the State of South Carolina, Charleston
OPHTHALMOLOGY—See also Otolaryngology					
George Washington University School of Medicine	January 26	6 days (surgery, pathology, orthoptics)	30	$100	Professor of Ophthalmology, George Washington University School of Medicine, 1325 H St., N.W., Washington, D. C.
	February 2	3 days	.....	$35	
University of Illinois College of Medicine	2d semester	4 months (pathology of the eye)	.....	$75 plus cost of materials	Assistant to the Dean, University of Illinois College of Medicine, 1853 W. Polk St., Chicago
Harvard Medical School, Courses for Graduates	January 5; on arrangement	2 weeks, mornings or 1 month, afternoons	Limited	$60 [9]	Assistant Dean, Harvard Medical School, 25 Shattuck St., Boston
	Arranged on application	3 months, daily (refraction)	Limited	$300 [9]	
	February 16	2 weeks (clinico-pathologic)	20	$75 [9]	
	March 2	2 weeks, mornings (neuro-ophthalmology)	8	$40 [9]	
	March 23	4 weeks, 3 mornings a week (ophthalmoscopy)	Limited	$60 [9]	
		4 weeks, 3 mornings a week (clinical)	Limited	$50 [9]	
	April 20	4 weeks, part time courses	8	$40 [9]	
Tufts Medical School, Postgraduate Division	February 2	4 weeks, 4 mornings a week (ophthalmoscopy)	3	$50 [9]	Chairman, Postgraduate Division, Tufts Medical School, 20 Bennet St., Boston
	March 2 [9]	3 months, mornings (refraction)	.....	$150 [9]	
	Monthly	3 mornings a week	Limited	$50 [9]	
Faculty of Medicine of Columbia University	Arranged on application [9]	15 sessions (embryology, histology, pathology)	1 or more	$75	Dean of the School of Medicine, Columbia University, 630 W. 168th St., New York City
	March 2 [9]	4 successive courses of 5-6 days	35	$85 to $90 a course	

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OPHTHALMOLOGY—See also Otolaryngology—Continued					
Graduate School of Medicine, University of Pennsylvania	Arranged on application	2 or 3 weeks (operations on cadaver) 8 weeks (refraction) 8 weeks (histology and pathology)	....	$100 to $270 $270 $330	Dean, Graduate School of Medicine, University of Pennsylvania, Philadelphia
ORTHOPEDIC SURGERY					
Denver General Hospital (Colorado State Medical Society)	February 16	3 days	35	$10	Executive Secretary, Colorado State Medical Society, 537 Republic Bldg., Denver
Harvard Medical School, Courses for Graduates	Monthly; April 1	1 month, mornings (clinical); 1 month full time	1 or more; 8 or more	$50; $150 [2]	Assistant Dean, Harvard Medical School, 25 Shattuck St., Boston
Tufts Medical School, Postgraduate Division	March 2	6 days	.....	$25 [2]	Chairman, Postgraduate Division, Tufts Medical School, 30 Bennet St., Boston
Faculty of Medicine of Columbia University	January 19 [2]	9 days (seminar)	5-20	$75	Dean of the School of Medicine, Columbia University, 630 W. 168th St., New York City
	March 23; April 6	6 days intensive; 4 weeks, 3 afternoons	4-20	$35; $50	
	April 6	1 month, 2 mornings a week (anatomy)	6-15	$40	
New York Polyclinic Medical School and Hospital	Arranged on application	3 months	.....	$75 to $100	Medical Executive Officer, New York Polyclinic Medical School, 345 W. 50th St., New York City
OTOLARYNGOLOGY					
The Research Study Club of Los Angeles	January 19	2 weeks (includes ophthalmology)	.....	$50	Secretary, The Research Study Club of Los Angeles, 2309 W. Washington Blvd., Los Angeles
	January 19	2 weeks, part time (anatomy of head and neck)	.....	$50	
College of Medical Evangelists.....	March 29	20 hours	6-10	$40	Chairman, Committee on Postgraduate Education, 312 N. Boyle Ave., Los Angeles
University of Illinois College of Medicine	January 19	4 months or more	.....	$75 a semester	Assistant to the Dean, University of Illinois College of Medicine, 1853 W. Polk St., Chicago
Tulane University of Louisiana Department of Graduate Medicine	March	4 weeks (preparation for board examinations)	20	Not available	Director, Department of Graduate Medicine, Tulane University of Louisiana, 1430 Tulane Ave., New Orleans
Harvard Medical School, Courses for Graduates	April	1 month (otology)	Limited	$185 [2]	Assistant Dean, Harvard Medical School, 25 Shattuck St., Boston
	Arranged on application	2 weeks (physiology); 1 month (clinical otology)	2	$50 [2]	
Tufts Medical School, Postgraduate Division	Monthly	1 month, mornings	.....	$50 [2]	Chairman, Postgraduate Division, Tufts Medical School, 30 Bennet St., Boston
University of Michigan Department of Postgraduate Medicine	April 16	1 week (includes ophthalmology)	.....	$10-$25	Chairman, Department of Postgraduate Medicine, University of Michigan, Ann Arbor
Center for Continuation Study, University of Minnesota	March 2 [2]	6 days	.....	$25	Center for Continuation Study, University of Minnesota, Minneapolis
Faculty of Medicine of Columbia University	Arranged on application	4 weeks, 3 afternoons a week	6	$30	Dean of the School of Medicine, Columbia University, 630 W. 168th St., New York City
	Arranged on application [2]	3 weeks (bronchoscopy) 4 courses in cadaver surgery	 2-6	$250 Varies	
New York Eye and Ear Infirmary...	March	1 week (includes ophthalmology)	Limited	$50	Registrar, New York Eye and Ear Infirmary, 218 Second Ave., New York City
	Arranged on application	1 to 3 months (includes ophthalmology)	.....	$40 to $110	
New York Polyclinic Medical School and Hospital	Arranged on application	6 weeks to 3 months (includes ophthalmology)	.....	$100 to $400	Medical Executive Officer, New York Polyclinic Medical School, 345 W. 50th St., New York City
University of Oregon Medical School, Oregon Academy of Ophthalmology and Otolaryngology	April 6 [2]	1 week (includes ophthalmology)	.....	$25	Dean, University of Oregon Medical School, 3181 S.W. Marquam Hill Rd., Portland
Gill Memorial Eye, Ear, Nose and Throat Hospital, Roanoke, Va.	April 6	1 week (includes ophthalmology)	50	$50	Medical Director, Gill Memorial Eye, Ear, Nose and Throat Hospital, 711 S. Jefferson St., Roanoke, Va.
PATHOLOGY					
Harvard Medical School, Courses for Graduates	Monthly	1 month	4	$40 [2]	Assistant Dean, Harvard Medical School, 25 Shattuck St., Boston
	Monthly [2]	1 month (obstetrics and gynecology)	2	$125 [2]	
Faculty of Medicine of Columbia University	January to April	Various part time courses	Limited	$45 to $125	Dean of the School of Medicine, Columbia University, 630 W. 168th St., New York City
PEDIATRICS—See also Obstetrics					
University of California School of Medicine	January 5	2 weeks	Limited	$20 [2]	Director of Maternal and Child Health in state health departments of California, Arizona, Nevada, New Mexico, Utah and Idaho
University of Illinois College of Medicine	Arranged on application	8 lectures, 4 months clinical (child psychology)	.....	Varies	Assistant to the Dean, University of Illinois College of Medicine, 1853 W. Polk St., Chicago
Tufts Medical School, Postgraduate Division	January 5	4 weeks	4	$50 [2]	Chairman, Postgraduate Division, Tufts Medical School, 30 Bennet St., Boston
University of Michigan Department of Postgraduate Medicine	April	1 week	.....	$10 to $25	Chairman, Department of Postgraduate Medicine, University of Michigan, Ann Arbor

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PEDIATRICS—See also Obstetrics—Continued					
Jewish Hospital, Brooklyn (Joint Committee on Postgraduate Education)	April	5 weeks, 3 times a week	6	$25	Registrar, Joint Committee on Post-Graduate Education, 1313 Bedford Ave., Brooklyn
Beth-El Hospital, Brooklyn (Joint Committee on Postgraduate Education)	April 6	5 weeks, twice a week	8	$10	
Faculty of Medicine of Columbia University	Monthly	1 month or longer (various courses)	Limited	$40 to $125	Dean of the School of Medicine, Columbia University, 630 W. 168th St., New York City
	March 9	5 days (recent advances)	5 or more	$35	
PERIPHERAL VASCULAR DISEASES					
The Mayo Foundation, University of Minnesota and The Mayo Clinic, Rochester	April 6	2 weeks	25-100 [1]	$40	Executive Secretary, American College of Physicians, 4200 Pine St., Philadelphia
Israel Zion Hospital, Brooklyn (Joint Committe on Postgraduate Education)	January 10	6 weeks, twice a week	8	$10	Registrar, Joint Committee on Post-Graduate Education, 1313 Bedford Ave., Brooklyn
PHYSICAL THERAPY					
Faculty of Medicine of Columbia University	April 6	5 days	4 or more	$35	Dean of the School of Medicine, Columbia University, 630 W. 168th St., New York City
New York Polyclinic Medical School and Hospital	Arranged on application	4 weeks or more	.....	$100	Medical Executive Officer, New York Polyclinic Medical School, 335 W. 50th St., New York City
PNEUMONIA					
University of Colorado School of Medicine (Colorado State Medical Society)	February 16	3 days	35	$10	Executive Secretary, Colorado State Medical Society, 537 Republic Bldg., Denver
State Hygienic Laboratory, Iowa City	Arranged on application	3 days (pneumonia typing technic)	.....	None [2]	Director, State Hygienic Laboratory, Iowa City
New York University College of Medicine	Arranged on application	4 weeks	4	$150	Assistant Dean, New York University College of Medicine, 477 First Ave., New York City
PROCTOLOGY					
Harvard Medical School, Courses for Graduates	April 13	6 days	8 or more	$50 [2]	Assistant Dean, Harvard Medical School, 25 Shattuck St., Boston
Tufts Medical School, Postgraduate Division	April 27	6 days	.....	$25 [2]	Chairman, Postgraduate Division, Tufts Medical School, 30 Bennet St., Boston
University of Michigan Department of Postgraduate Medicine	April	5 days	.....	$10-$25	Chairman, Department of Postgraduate Medicine, University of Michigan, Ann Arbor
Brooklyn Hospital (Joint Committee on Post-Graduate Education)	February 24	3 weeks, 4 mornings a week	4	$10	Registrar, Joint Committee on Post-Graduate Education, 1313 Bedford Ave., Brooklyn
Jewish Hospital, Brooklyn (Joint Committee on Post-Graduate Education)	April 1	3 weeks, 3 mornings a week	8	$25	
Faculty of Medicine of Columbia University	January 2 [3]	3 months, 3 afternoons a week	2-4	$150	Dean of the School of Medicine, Columbia University, 630 W. 168th St., New York City
New York Polyclinic Medical School and Hospital	January 2; April 1	6 weeks (various courses)	.....	$75 to $100	Medical Executive Officer, New York Polyclinic Medical School, 335 W. 50th St., New York City
PUBLIC HEALTH					
Loyola University School of Medicine	February 2	4 month semester (various courses)	.....	$10 to $125	Dean, Loyola University School of Medicine, 706 S. Wolcott Ave., Chicago
Johns Hopkins University School of Hygiene and Public Health	January 24; March 21	8 week quarter (various courses)	.....	$10 to $75	Dean, School of Hygiene and Public Health, Johns Hopkins University, 615 N. Wolfe St., Baltimore
Harvard Medical School, Courses for Graduates	February	4 months, hours arranged (epidemiology)	.....	Arranged	Assistant Dean, Harvard Medical School, 25 Shattuck St., Boston
Harvard School of Public Health	Arranged on application	1 month or more	.....	Varies	Dean, School of Public Health, 55 Shattuck St., Boston
Massachusetts Institute of Technology	2d semester	5 months	.....	$300 a semester	Admissions Office, Massachusetts Institute of Technology, Cambridge
Albany Medical College	Arranged on application	1 year correspondence course, 11 conferences, 2 days in residence	.....	$30	Director, Extension Course in Public Health, Albany Medical College, New Scotland Ave., Albany, N. Y.
Postgraduate Institute on Public Health, Tuberculosis League of Pittsburgh	April	4 days (institute for Negro physicians)	.....	No	Tuberculosis League of Pittsburgh, 2851 Bedford Ave., Pittsburgh
RADIOLOGY					
Harvard Medical School, Courses for Graduates	Monthly	1 month, 3 or 5 days a week (general and special roentgenology)	Limited	$35 to $100 [2]	Assistant Dean, Harvard Medical School, 25 Shattuck St., Boston
	April	1 month, 2 evenings a week (physics)	.....	$10 [2]	
Tufts Medical School, Postgraduate Division	January 13	4 days	.....	$25 [2]	Chairman, Postgraduate Division, Tufts Medical School, 30 Bennet St., Boston
University of Michigan Department of Postgraduate Medicine	April	1 week	.....	$10-$25	Chairman, Department of Postgraduate Medicine, University of Michigan, Ann Arbor
Faculty of Medicine of Columbia University	January 5 [3]	3 months, afternoons	.....	$125	Dean of the School of Medicine, Columbia University, 630 W. 168th St., New York City

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RADIOLOGY—Continued					
New York Polyclinic Medical School and Hospital	First of any month	6 week and 3 month courses (interpretation and technic of roentgenology)	.....	$150 to $300	Medical Executive Officer, New York Polyclinic Medical School, 335 W. 50th St., New York City
New York University College of Medicine	Spring	3 months, 3 afternoons a week	.....	$100	Office of the Dean, New York University College of Medicine, 477 First Ave., New York City
SURGERY					
Tulane University of Louisana Department of Graduate Medicine	January 5; February 16	6 week courses	.....	$150 to $225	Director, Department of Graduate Medicine, Tulane University of Louisiana, 1430 Tulane Ave., New Orleans
Harvard Medical School, Courses for Graduates	February 2	4 weeks	Limited	$100 [2]	Assistant Dean, Harvard Medical School, 25 Shattuck St., Boston
Center for Continuation Study, University of Minnesota	January 19	6 days (emergency surgery in war time)	.....	$25	Center for Continuation Study, University of Minnesota, Minneapolis
Cumberland Hospital, Brooklyn (Joint Committee on Post-Graduate Education)	April 6	4 weeks, 2 afternoons a week (traumatic)	8	$10	Registrar, Joint Committee on Post-Graduate Education, 1313 Bedford Ave., Brooklyn
Faculty of Medicine of Columbia University	March 16; April 20 [4]	6 days (traumatic)	5-30	$35; $60	Dean of the School of Medicine, Columbia University, 630 W. 168th St., New York City
	April 6 [3]	6 week or 3 month seminar course	.....	$200 to $400	
	Arranged on application	12 sessions in surgical anatomy; 8 in blood transfusion	Limited	$75 to $200; $35	
New York Medical College.....	Arranged on application	60 hours (experimental surgery and technic)	1-4	$250 for single student	Dean, New York Medical College, 5th Ave. at 105th St., New York City
New York Polyclinic Medical School and Hospital	January 2; April 1	3 months	.....	$350	Medical Executive Officer, New York Polyclinic Medical School, 335 W. 50th St., New York City
	Arranged on application	6 weeks	.....	$100	
TUBERCULOSIS					
University of Colorado School of Medicine	April 13	6 days	10-25 [1]	$20	Executive Secretary, American College of Physicians, 4200 Pine St., Philadelphia
Mississippi State Tuberculosis Sanatorium	Arranged on application	2 weeks or more	.....	None	Medical Director, State Sanatorium, Sanatorium, Miss.
Faculty of Medicine of Columbia University	February 2	2 weeks (diseases of the chest)	4-12	$50	Dean of the School of Medicine, Columbia University, 630 W. 168th St., New York City
Cornell University Medical College	February 11; April 8	1 day	.....	None	Secretary, Tuberculosis Sanatorium Conference of Metropolitan New York, 386 Fourth Ave., New York City
New York Medical College.....	Arranged on application	1 month	.....	$100	Dean, New York Medical College, 5th Ave. at 105th St., New York City
UROLOGY					
College of Medical Evangelists.....	January 4; March 29	1 month, 50 hours	2-4	$100	Chairman, Committee on Postgraduate Education, 312 N. Boyle Ave., Los Angeles
Graduate School of Medicine, University of Pennsylvania	Arranged on application	6 weeks, 36 hours (cystoscopy, etc.)	.....	$300	Dean, Graduate School of Medicine, University of Pennsylvania, 36th and Pine Sts., Philadelphia
Harvard Medical School, Courses for Graduates	April	1 month, mornings (genito-urinary surgery)	4	$75 [2]	Assistant Dean, Harvard Medical School, 25 Shattuck St., Boston
Tufts Medical School, Postgraduate Division	April 13	6 days	.....	$25	Chairman, Postgraduate Division, Tufts Medical School, 30 Bennet St., Boston
Long Island College Hospital, Brooklyn (Joint Committee on Post-Graduate Education)	1st of each month	1 month or more	3	$25 a month	Registrar, Joint Committee on Post-Graduate Education, 1313 Bedford Ave., Brooklyn
VENEREAL DISEASES					
City Hospital, Mobile, Ala.....	3d week of month	1 week	Limited	None [5]	Director, Division of Venereal Disease Control, State Health Department, Montgomery, Ala.
Clinic at Hot Springs National Park, Ark.	April	4 weeks (venereal disease control)	Limited	None [5]	Medical officer in charge, U. S. P. H. S. Medical Center, Hot Springs, Ark.
Howard University College of Medicine	January 3; March 21	3 months (venereal disease control)	10	$20 [6,7]	Dean, Howard University College of Medicine, Washington, D. C.
Harvard Medical School, Courses for Graduates	Monthly	10 sessions (gonorrhea in women)	.....	$20 [2]	Assistant Dean, Harvard Medical School, 25 Shattuck St., Boston
Faculty of Medicine of Columbia University	Arranged on application	6 week and 3 month courses, part time	.....	$40 and $75	Dean of the School of Medicine, Columbia University, 630 W. 168th St., New York City
New York Polyclinic Medical School and Hospital	Arranged on application	6 week and 3 month courses	.....	$35 to $75	Medical Executive Officer, New York Polyclinic Medical School, 335 W. 50th St., New York City
Institute for the Control of Syphilis, Hospital of the University of Pennsylvania	Arranged on application [8]	1 or 2 weeks; 1 month (venereal disease control)	12	$25; $50	Director, Institute for the Control of Syphilis, Hospital of the University of Pennsylvania, 34th and Spruce St., Philadelphia

A limited number of opportunities for postgraduate study in the clinical departments are also offered by the University of Chicago, The School of Medicine; Tulane University of Louisiana School of Medicine; Johns Hopkins University School of Medicine; Duke University School of Medicine; Vanderbilt University School of Medicine; Meharry Medical College, and the University of Wisconsin Medical School.

1. The courses are organized especially for Fellows and Associates of the American College of Physicians, but where facilities are available, courses will be open to those with adequate preliminary training who are now preparing either to meet the requirements of membership in the College or certification by the American Board of Internal Medicine.
2. Registration fee of $5 covers all courses taken within twelve months.
3. Admission is limited to physicians who have had adequate previous training and experience in the special field.
4. A faculty course given for staff members of mental institutions.
5. State boards of health furnish either the funds covering tuition fees, maintenance or transportation for physicians of the state.
6. Trainees are usually recommended by state or city health departments.
7. Applicants may qualify for a monthly stipend from the United States Public Health Service.
8. Short course scheduled on arrangement of health department or agency; one month course on an individual basis.

Medical Examinations and Licensure

COMING EXAMINATIONS AND MEETINGS

ANNUAL CONGRESS ON MEDICAL EDUCATION AND LICENSURE

CHICAGO, Feb. 16-17, 1942. Council on Medical Education and Hospitals, Sec., Dr. William D. Cutter, 535 North Dearborn Street, Chicago.

MEDICAL CORPS, UNITED STATES NAVY

*Examination.** Assistant Surgeon with the permanent rank of Lieutenant (junior grade) and Acting Assistant Surgeon with the probationary rank of Lieutenant (junior grade), Jan. 5-9. Examination will be held at the Naval Hospitals at Chelsea, Mass., Newport, R. I., Brooklyn, Philadelphia, Norfolk, Va., Charleston, S. C., Pensacola, Fla., Corpus Christi, Tex., San Diego and Mare Island, Calif., Puget Sound, Wash., Great Lakes, Ill., Pearl Harbor, T. H., and Naval Medical Center, Washington, D. C. Apply Bureau of Medicine and Surgery, Navy Department, Washington, D. C.

NATIONAL BOARD OF MEDICAL EXAMINERS EXAMINING BOARDS IN SPECIALTIES

Examinations of the National Board of Medical Examiners and Examining Boards in Specialties were published in THE JOURNAL, December 27, page 2272.

BOARDS OF MEDICAL EXAMINERS

ALABAMA: Montgomery, June 16-18. Sec., Dr. B. F. Austin, 519 Dexter Ave., Montgomery.

ARIZONA: * Phoenix, Jan. 6-7. Sec., Dr. J. H. Patterson, 826 Security Bldg., Phoenix.

ARKANSAS: * Little Rock, June 4-5. Sec., Dr. D. L. Owens, Harrison.

CONNECTICUT: * *Medical Examination.* Hartford, March 10-11. *Endorsement.* Hartford, March 24. Sec. to the Board, Dr. Creighton Barker, 258 Church St., New Haven. *Homeopathic.* Derby, March 10-11. Sec., Dr. Joseph H. Evans, 1488 Chapel St., New Haven.

DELAWARE: Dover, July 14-16. Sec., Medical Council of Delaware, Dr. Joseph S. McDaniel, 229 S. State St., Dover.

FLORIDA: * Jacksonville, June 22-23. Sec., Dr. William M. Rowlett, Box 786, Tampa.

GEORGIA: Atlanta, June. Sec., State Examining Boards, Mr. R. C. Coleman, 111 State Capitol, Atlanta.

HAWAII: Honolulu, Jan. 12-13. Sec., Dr. James A. Morgan, 48 Young Bldg., Honolulu.

IDAHO: Boise, Jan. 13. Dir., Bureau of Occupational License, Mr. Walter Curtis, 355 State Capitol Bldg., Boise.

ILLINOIS: Chicago, Jan. 20-22. Superintendent of Registration, Mr. Philip M. Harman, Department of Registration and Education, Springfield.

INDIANA: Indianapolis, June 16-18. Sec., Board of Registration and Examination, Dr. J. W. Bowers, 301 State House, Indianapolis.

IOWA: * Des Moines, Jan. 15-17. Dir., Division of Licensure and Registration, State Department of Health, Mr. H. W. Grefe, Capitol Bldg., Des Moines.

MAINE: Portland, March 10-11. Sec., Board of Registration of Medicine, Dr. Adam P. Leighton, 192 State St., Portland.

MASSACHUSETTS: Boston, March 10-13. Sec., Board of Registration in Medicine, Dr. Stephen Rushmore, 413-F State House, Boston.

MICHIGAN: * Ann Arbor and Detroit, June 10-12. Sec., Board of Registration in Medicine, Dr. J. Earl McIntyre, 202-4 Hollister Bldg., Lansing.

MINNESOTA: * Minneapolis, Jan. 20-22. Sec., Dr. Julian F. Du Bois, 230 Lowry Medical Arts Bldg., St. Paul.

MONTANA: Helena, April 7-8. Sec., Dr. Otto G. Klein, First National Bank Bldg., Helena.

NEVADA: *Reciprocity.* Carson City, February 2. Sec., Dr. Frederick M. Anderson, 215 N. Carson Street, Carson City.

NEW HAMPSHIRE: Concord, March 12-13. Sec., Dr. T. P. Burroughs, Board of Registration in Medicine, State House, Concord.

NEW JERSEY: Trenton, June 16-17. Sec., Dr. Earl S. Hallinger, 28 W. State St., Trenton.

NEW MEXICO: * Santa Fe, April 13-14. Sec., Dr. Le Grand Ward 135 Sena Plaza, Santa Fe.

NEW YORK: Albany, Buffalo, New York and Syracuse, Jan. 26-29. Chief, Bureau of Professional Examinations, Mr. Herbert J. Hamilton, State Education Department, 315 Education Bldg., Albany.

NORTH DAKOTA: Grand Forks, Jan. 6-9. Sec., Dr. G. M. Williamson, 455 S. Third St., Grand Forks.

OREGON: Portland, Jan. 21-23. Exec. Sec., Miss Lorienne M. Conlee, 608 Failing Bldg., Portland.

PENNSYLVANIA: Philadelphia, Jan. 6-10. Acting Sec., Bureau of Professional Licensing, Mrs. Marguerite G. Steiner, 358 Education Bldg., Harrisburg.

RHODE ISLAND: * Providence, Jan. 8-9. Chief, Division of Examiners, Mr. Thomas B. Casey, 366 State Office Bldg., Providence.

SOUTH DAKOTA: * Pierre, Jan. 13-14. Dir., Medical Licensure, Dr. J. F. D. Cook, State Board of Health, Pierre.

TEXAS: Galveston, March 23-25. Sec., Dr. T. J. Crowe, 918-20 Texas Bank Bldg., Dallas.

VERMONT: Burlington, Feb. 10-12. Sec., Board of Medical Registration, Dr. F. J. Lawliss, Richford.

WEST VIRGINIA: Charleston, March 2-4. Commissioner, Public Health Council, Dr. C. F. McClintic, State Capitol, Charleston.

WASHINGTON: * Seattle, Jan. 12-14. Dir., Department of Licenses, Mr. Thomas A. Swayze, Olympia.

WISCONSIN: * Madison, Jan. 13-15. Sec., Dr. H. W. Shutter, 425 E. Wisconsin Ave., Milwaukee.

WYOMING: Cheyenne, Feb. 2-3. Sec., Dr. M. C. Keith, Capitol Bldg., Cheyenne.

* Basic Science Certificate required.

BOARDS OF EXAMINERS IN THE BASIC SCIENCES

CONNECTICUT: Feb. 14. Address State Board of Healing Arts, 1945 Yale Station, New Haven.

DISTRICT OF COLUMBIA: Washington, April 20-21. Sec., Commission on Licensure, Dr. George C. Ruhland, 6150 E. Municipal Bldg., Washington.

FLORIDA: Gainesville, June 8. Sec., Professor J. F. Conn, John B. Stetson University, De Land.

IOWA: Des Moines, Jan. 13. Dir., Division of Licensure and Registration, State Department of Health, Mr. H. W. Grefe, Capitol Bldg., Des Moines.

MICHIGAN: February 13-14. Sec., Miss Flora E. Dube, East Lansing.

MINNESOTA: Minneapolis, Jan. 6-7. Sec., Dr. J. Charnley McKinley, 126 Millard Hall, University of Minnesota, Minneapolis.

NEBRASKA: Omaha, Jan. 13-14. Dir., Bureau of Examining Boards, Mrs. Jeannette Crawford, 1009 State Capitol Bldg., Lincoln.

NEW MEXICO: Albuquerque, Feb. 2. Sec., Miss Pia Joerger, State Capitol, Santa Fe.

OREGON: Portland, Feb. 14. Applications must be on file not later than than Jan. 28. Sec., State Board of Higher Education, Mr. Charles D. Byrne, University of Oregon, Eugene.

RHODE ISLAND: Providence, Feb. 18. Chief, Division of Examiners, Mr. Thomas B. Casey, 366 State Office Bldg., Providence.

WASHINGTON, Seattle, Jan. 8-9. Dir., Department of Licenses, Mr. Thomas A. Swayze, Olympia.

Texas June Report

The Texas State Board of Medical Examiners reports the written examination for medical licensure held at Austin, June 16-18, 1941. The examination covered 12 subjects and included 120 questions. An average of 75 per cent was required to pass. One hundred and seventy-one candidates were examined, 162 of whom passed and 9 failed. Sixty-one physicians were licensed to practice medicine by reciprocity and 2 physicians so licensed by endorsement. The following schools were represented:

PASSED	Year Grad.	Number Passed
Rush Medical College	(1939)	1
Tulane University of Louisiana School of Medicine	(1941)	1
Washington University School of Medicine	(1940)	1
University of Oklahoma School of Medicine	(1939), (1941)	2
Temple University School of Medicine	(1940)	1
University of Pennsylvania School of Medicine	(1940), (1941)	2
Baylor University College of Medicine	(1941, 62)	62
University of Texas School of Medicine	(1941, 83)	83
University of Wisconsin Medical School	(1940, 2)	2
Osteopaths *		7

FAILED	Year Grad.	Number Failed
Baylor University College of Medicine	(1941)	1
University of Texas School of Medicine	(1941, 3)	3
Osteopaths *		5

LICENSED BY RECIPROCITY	Year Grad.	Reciprocity with
College of Medical Evangelists	(1938)	California
University of Colorado School of Medicine	(1928)	Indiana
Georgetown University School of Medicine	(1938)	Maine
Howard University College of Medicine	(1937)	Virginia
University of Georgia School of Medicine	(1925)	Georgia
The School of Medicine of the Division of Biological Sciences	(1938)	Minnesota
University of Chicago, The School of Medicine	(1939)	Ohio
Univ. of Illinois College of Medicine	(1930), (1936), (1940)	Illinois
Central College of Physicians and Surgeons, Indianapolis	(1900)	Indiana
Physio-Medical College of Indiana	(1898)	Oklahoma
Indiana University School of Medicine	(1937)	Indiana
State University of Iowa College of Medicine	(1938)	Iowa
Kansas Medical College, Medical Department of Washburn College	(1900)	Oklahoma
University of Kansas School of Medicine	(1919), (1936)	Kansas
University of Louisville School of Medicine	(1926)	Kentucky
Louisiana State University Medical Center	(1934)	Louisiana
Louisiana State University School of Medicine	(1940)	Louisiana
Tulane University of Louisiana School of Medicine	(1925), (1936), (1938, 2), (1939), (1940, 2)	Louisiana
Harvard Medical School	(1936)	New York, (1938) Connecticut
University of Michigan Medical School	(1933)	Michigan
Wayne University College of Medicine	(1941)	Michigan
University of Nebraska College of Medicine	(1924), (1937), (1938)	Nebraska
Bellevue Hospital Medical College	(1898)	New York
Columbia University College of Physicians and Surgeons	(1931), (1935)	New York
Ohio State University College of Medicine	(1927)	Ohio
University of Cincinnati College of Medicine	(1930)	Kentucky, (1940) Ohio
Western Reserve University Medical Department	(1912)	Ohio
Western Reserve University School of Medicine	(1926), (1938)	Ohio
University of Oklahoma School of Medicine	(1917), (1931), (1933, 2), (1939)	Oklahoma
University of Pittsburgh School of Medicine	(1930)	Penna.
Medical College of the State of South Carolina	(1940)	S. Carolina
Meharry Medical College	(1927)	Louisiana, (1938), (1940, 3) Tennessee
University of Tennessee College of Medicine	(1927)	New York, (1935), (1938) Tennessee
Marquette University School of Medicine	(1925), (1937) (1940)	Wisconsin
University of Wisconsin Medical School	(1938)	Wisconsin

LICENSED BY ENDORSEMENT	Year Grad.	Endorsement of
University of Illinois College of Medicine	(1930)	U. S. Navy
Jefferson Medical College of Philadelphia	(1931)	U. S. Navy

* Examined in medicine and surgery.

Wisconsin June Report

The Wisconsin State Board of Medical Examiners reports the written examination for medical licensure held at Milwaukee, June 24-26, 1941. The examination covered 19 subjects and included 100 questions. An average of 75 per cent was required to pass. One hundred and four candidates were examined, all of whom passed. Twenty-eight physicians were licensed to practice medicine by reciprocity. The following schools were represented:

School	PASSED Year Grad.	Number Passed
College of Medical Evangelists	(1941)	1
Loyola University School of Medicine	(1941)	1
Northwestern University Medical School	(1940, 2), (1941, 4)	6
Rush Medical College	(1940, 4)	4
University of Illinois College of Medicine	(1941, 7)	7
University of Louisville School of Medicine	(1940)	1
University of Michigan Medical School	(1940)	1
University of Minnesota Medical School	(1940), (1941, 2)*	3
University of Nebraska College of Medicine	(1940)	1
Hahnemann Medical College and Hospital of Philadelphia	(1940)	1
Temple University School of Medicine	(1940, 2)	2
Medical College of Virginia	(1940)	1
Marquette University School of Medicine	(1941, 47)	47
Univ. of Wisconsin Med. School	(1938), (1939),* (1940, 24)	26
University of Toronto Faculty of Medicine	(1936)	1
Osteopaths †		2

School	LICENSED BY RECIPROCITY Year Grad.	Reciprocity with
University of Arkansas School of Medicine	(1932)	Arkansas
University of California Medical School	(1932), (1933)	California
Loyola University School of Medicine	(1940)	Illinois
Northwestern University Medical School	(1925) (1937), (1940)	North Dakota, Illinois
Rush Medical College	(1928) Michigan, (1936), (1939, 2)	Illinois
University of Chicago, The School of Medicine	(1939)	Minnesota
University of Illinois College of Medicine	(1939)	Illinois
University of Kansas School of Medicine	(1939)	Kansas
Indiana University School of Medicine	(1939)	Indiana
University of Louisville School of Medicine	(1937)	Kentucky
University of Michigan Medical School	(1939)	Michigan
Wayne University College of Medicine	(1936)	Michigan
University of Minnesota Medical School	(1939), (1940, 2)	Minnesota
Washington University School of Medicine	(1939, 2)	Missouri
Creighton University School of Medicine	(1930)	Nebraska
Cornell University Medical College	(1938)	New York
Long Island College of Medicine	(1939)	New York
University of Wisconsin Medical School	(1933) (1938) Louisiana	Oklahoma,

* Licenses have not been issued.
† Examined in surgery.

Montana October Report

The Montana State Board of Medical Examiners reports the written examination for medical licensure held at Helena, Oct. 6-8, 1941. The examination covered 10 subjects. An average of 75 per cent was required to pass. Two candidates were examined, both of whom passed. Eight physicians were licensed to practice medicine by reciprocity and 2 physicians so licensed on endorsement of credentials of the National Board of Medical Examiners. The following schools were represented:

School	PASSED Year Grad.	Number Passed
George Washington University School of Medicine	(1910)	1
Université de Lausanne Faculté de Médecine	(1937)	1

School	LICENSED BY RECIPROCITY Year Grad.	Reciprocity with
University of Colorado School of Medicine	(1940)	Colorado
Northwestern University Medical School	(1938)	Indiana
Tulane University of Louisiana School of Medicine	(1935)	Minnesota
University of Minnesota Medical School	(1935), (1937, 2)	Minnesota
St. Louis University School of Medicine	(1941)	Missouri
Marquette University School of Medicine	(1935)	Wisconsin

School	LICENSED BY ENDORSEMENT Year Grad.
Northwestern University Medical School	(1937)
Creighton University School of Medicine	(1940)

Oregon July Report

The Oregon State Board of Medical Examiners reports the written examination for medical licensure held at Portland, July 24-26, 1941. The examination covered 12 subjects and included 86 questions. An average of 75 per cent was required to pass. Nineteen candidates were examined, all of whom passed. The following schools were represented:

School	PASSED Year Grad.	Number Passed
College of Medical Evangelists	(1941, 5)	5
University of Illinois College of Medicine	(1941)	1
University of Kansas School of Medicine	(1939), (1940)	2
University of Nebraska College of Medicine	(1937)	1
University of Oregon Medical School	(1939), (1940, 8)	9
Osteopath *		1

* Examined in surgery.

Bureau of Legal Medicine and Legislation

MEDICOLEGAL ABSTRACTS

Malpractice: Fraudulent Concealment as Affecting Statute of Limitations.—In March 1929 the defendant dentist removed an impacted wisdom tooth, and an attached pus sac, from the plaintiff's lower left jaw and a few months later also extracted her lower right wisdom tooth. She continued to have headaches and pain in various parts of her body, however, and consulted the defendant again the latter part of 1929 but was advised that she was not suffering from any infection due to her teeth. During the next several years the plaintiff consulted many doctors and submitted to operations for the removal of her tonsils, appendix and gallbladder, but the pain in her face continued. In March 1938 a roentgenogram taken by some dentist other than the defendant revealed two fragments of tooth root in the plaintiff's lower left jaw bone. These were removed and the plaintiff gradually regained her health. In a subsequent suit against the defendant for malpractice, the jury returned a verdict for the defendant, and the plaintiff appealed to the Supreme Court of Errors of Connecticut.

The plaintiff contended that the defendant had fraudulently concealed from her the existence of the broken pieces of tooth in her jaw and that therefore the defense of the statute of limitations was not available. The Supreme Court of Errors said that, in order for the plaintiff to sustain her contention, she was required to prove that the defendant had actual or constructive knowledge that the fragments of tooth remained in the plaintiff's jaw after the extraction. Without such knowledge there could be no fraudulent concealment. The plaintiff testified that the defendant showed her the tooth after it had been extracted and called her attention to the pus sac attached thereto. Nothing was then said or done by the defendant which indicated a knowledge that any part of the tooth still remained in her jaw. Furthermore, there was no expert testimony supporting the theory that, in the exercise of due care and skill, the defendant should have known that the fragment remained. The court therefore held that there was nothing in the record to indicate a finding that the defendant either knew or should have known of such condition or that he was guilty of a gross want of due care. The court further held that the plaintiff could not rely on the doctrine of res ipsa loquitur. Such doctrine could be applied to infer negligence only when there were some facts proved which afforded a basis for such inference. Here there were no such facts shown. Applied to this case, therefore, the doctrine would only infer negligence in failing to discover that the fragments remained rather than an actual knowledge thereof. The judgment for the defendant was therefore affirmed.—*Frogge v. Shugrue, 13 A. (2d) 503 (Conn., 1940).*

Medical Practice Acts: Graduates from Foreign Medical Schools; Foreign Licensure Requirement.—The plaintiff, a citizen of the United States and a resident of the state of California, received one year of medical instruction at the University of Albert-Ludwig in Germany and then concluded his medical studies at the University of Basle in Switzerland. He then returned to the United States and served an eighteen months internship in the Los Angeles County General Hospital. The evidence showed that the medical schools which the plaintiff attended and the hospital in which he interned were all approved by the defendant board as proper places for instruction and internship. When the plaintiff subsequently petitioned the defendant, the Board of Medical Examiners of the State of California, for permission to take the regular examination for a physician's and surgeon's license, however, he was denied that right on the ground that he had failed to show that he was licensed to practice medicine in Switzerland. The plaintiff filed

a petition for a writ of mandamus to compel the board to permit him to take the examination. The trial court found in favor of the plaintiff and the defendant appealed to the district court of appeal, second district, division 2, California.

The Business and Professions Code of California (Section 2193) requires that a person whose application for examination is based on a diploma issued by a foreign medical school must show, among other things, that

> (c) He has been admitted or licensed to practice medicine and surgery in the country where the institution in which he has pursued his medical course of professional instruction is located.

The plaintiff contended that this requirement constituted an unreasonable, arbitrary and discriminatory exercise of the police power. It was necessary to determine, said the district court of appeal, whether or not the legislature intended the requirement to apply both to aliens and to American citizens. The court found that it was impossible for an American citizen to obtain a license to practice medicine in Switzerland and that, because of this inability, an alien applicant who had attended a foreign school was in a better position before the California board than was a citizen who had so attended, because the alien was able to obtain a license to practice in his native country. To the court it seemed unreasonable to believe that the legislature intended such an unjust result. The court therefore assumed that the legislature had been properly informed on the subject matter of the requirement and knew, at the time Section 2193 (*c*) was enacted, that American citizens were unable to obtain the required foreign license. It therefore concluded that the legislature must have meant the requirement to apply only to alien petitioners. Any other holding, said the court, would result in an injustice and an absurdity. The judgment for the plaintiff was accordingly affirmed.—*Lee v. Board of Medical Examiners, 97 P. (2d) 1046 (Calif., 1940).*

Malpractice: Liability of Physician for Negligence of Nurse.—One of the plaintiffs, Mrs. Kelly, took her infant son to the office of the defendant physician for an examination. While she was waiting her turn to see the doctor, a nurse employed by the defendant requested her to assist in holding a child on the x-ray table. The nurse-technician was in sole charge of the x-ray room and apparatus. In order to do this, Mrs. Kelly was directed to take a position between the x-ray table and the wall, touching both, with her hands on the child's ankles. She noticed that the child's ankles were wet but was advised that that was a matter of no importance. While Mrs. Kelly was thus acting as requested, the nurse turned on the electric current. Instantly there was a flash and a spark from the mechanism to Mrs. Kelly's head, the light in the x-ray machine immediately went out and Mrs. Kelly fell to the floor. She was both burned by the electric current and injured by the fall. Subsequently the plaintiffs, Mr. and Mrs. Kelly, sued the defendant physician for damages alleged to have been caused by the negligence of the defendant's nurse. From a judgment in favor of the plaintiffs, the defendant appealed to the Supreme Court of Pennsylvania.

The defendant contended that the judgment should be reversed because there was no evidence of any negligence chargeable to him. The Supreme Court said that when a physician burns a patient in the course of a roentgen treatment no prima facie liability attaches because burns do occur occasionally in the ordinary course of an exposure in spite of the highest degree of diligence and care to prevent them. In such a case the patient, in order to recover damages, must show that the apparatus was different from that ordinarily used or that the technician was incompetent to use it. The wife-plaintiff, however, continued the court, was not a patient of the defendant at the time she received her injuries. She was acting at the request and under the direction of the defendant's agent and thus did not assume the risks which a patient had to assume. When a thing which causes an injury is shown to be under the management of the defendant, the Supreme Court continued, and the accident is such as in the ordinary course of things does not happen if those who have the management use proper care, it affords reasonable evidence, in the absence of any explanation by the defendants, that the accident arose from such want of care. The effect of this principle is to require the defendant, in a case of this nature, to produce affirmative proof of his own freedom from fault. No such evidence was produced on behalf of the defendant, and the jury was justified, in the opinion of the court, in finding that the nurse was negligent.

The Supreme Court further found that the nurse-technician was the defendant's servant and that the taking of roentgenograms was within the scope of her employment. Moreover, her implied authority included the control of the patient as well as the actual operation of the apparatus. Her act in inducing the wife-plaintiff to assist her was thus incidental to the defendant's business and in furtherance of it, said the court, even though it was not specifically authorized. The defendant was therefore responsible for the consequences of her act. The judgment for the plaintiffs was accordingly affirmed.—*Kelly et al. v. Yount, 7 A. (2d) 582; 12 A. (2d) 579 (Penna., 1940).*

Workmen's Compensation Acts: Compensability for Death Due to Bacillus Enteritidis.—The duties of the employee consisted of skinning and butchering animals for use in the manufacture of chicken feed and other animal by-products. On Aug. 10 or 12, 1937, he became ill while at work and died twelve days later, death being attributed to Bacillus enteritidis. An award of compensation was denied by the industrial commission, and the widow of the employee appealed to the Supreme Court of Utah.

The workmen's compensation act of Utah provides that "(5) 'Personal injury by accident arising out of or in the course of employment' . . . shall not include a disease, except as it shall result from the injury." The defendant contended that the employee did not die as the result of an accident within the meaning of the act but rather as the result of a disease. The Supreme Court said that an accident is not limited in meaning to the application of physical force to the body of the injured but that infection by germs may be an accidental or unexpected event, even though not preceded by physical force. On the other hand, the Court continued, an illness is not compensable merely because it is contracted while the employee is engaged in his usual occupation; there still has to be a definite causal connection between the employment and the illness. The Court found that the disease from which the employee in this case died was rare and uncommon. It was contracted from diseased meat and the employee came in contact with such meat at the place he worked. The only reasonable conclusion, held the Supreme Court, was that the employee contracted the disease in the course of his employment. Judgment for the defendant was therefore reversed and the cause remanded for further proceedings.—*Andreason v. Industrial Commission, 100 P. (2d) 202; 102 P. (2d) 894 (Utah, 1940).*

Society Proceedings

COMING MEETINGS

Annual Congress on Industrial Health, Chicago, Jan. 12-13. Dr. C. M. Peterson, 535 North Dearborn St., Chicago, Secretary.

Annual Congress on Medical Education and Licensure, Chicago, Feb. 16-17. Dr. William D. Cutter, 535 North Dearborn St., Chicago, Secretary.

American Academy of Orthopedic Surgeons, Atlantic City, N. J., Jan. 11-15. Dr. Rexford L. Diveley, 1103 Grand Ave., Kansas City, Mo., Secretary.

American Orthopsychiatric Association, Detroit, Feb. 19-21. Dr. Norvelle C. LaMar, 149 East 73d St., New York, Secretary.

Central Surgical Association, Chicago, Feb. 27-28. Dr. George M. Curtis, Ohio State University, Columbus, Ohio.

Pacific Coast Surgical Association, San Francisco and Del Monte, Calif., Feb. 17-20. Dr. F. L. Reichert, Stanford University Hospital, San Francisco, Secretary.

Society of Surgeons of New Jersey, Trenton, Jan. 28. Dr. Walter B. Mount, 21 Plymouth St., Montclair, Secretary.

Current Medical Literature

AMERICAN

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Titles marked with an asterisk (*) are abstracted below.

American Journal of Medical Sciences, Philadelphia

202:469-624 (Oct.) 1941

Clinical Studies with Ballistocardiograph in Congestive Failure, on Digitalis Action, on Changes in Ballistic Form and in Certain Acute Experiments. I. Starr, Philadelphia.—p. 469.
Synovial Fluid and Synovial Membrane Abnormalities Resulting from Varying Grades of Systemic Infection and Edema. H. C. Coggeshall, G. A. Bennett, C. F. Warren and W. Bauer, Boston.—p. 486.
Studies of B Vitamins in Human Subject: I. Intake of Thiamine and Its Relation to Other Dietary Constituents in Food Selected by Normal Subject. K. O. Elsom and T. E. Machella, Philadelphia.—p. 502.
Id.: II. Urinary Excretion of Ingested Thiamine in Patients with Chronic Hepatic Disease. T. E. Machella and K. O. Elsom, with technical assistance of Charlotte S. Chornock, Philadelphia.—p. 512.
Protein Bound Iodine in Blood: V. Naturally Occurring Iodine Fractions and Their Chemical Behavior. A. M. Bassett, A. H. Coons and W. T. Salter, with technical assistance of Sophia M. Simmons, Boston.—p. 516.
Id.: VI. Its Relation to Thyroid Function in 100 Clinical Cases. W. T. Salter, A. M. Bassett and T. S. Sappington, with technical assistance of Sophia M. Simmons, Boston.—p. 527.
Experimental Aspiration Pneumonia: Fluorescence and Pathology. I. J. Wolman and Anna B. Bayard, Philadelphia.—p. 542.
*Action of Globin Insulin Compared with That of Crystalline, Unmodified, and Protamine Zinc Insulin. G. G. Duncan and C. E. Barnes, Philadelphia.—p. 553.
Studies on Hemorrhagic Agent 3,3'-Methylenebis (4-Hydroxycoumarin): I. Its Effect on Prothrombin and Coagulation Time of Blood of Dogs and Humans. J. B. Bingham, O. O. Meyer and F. J. Pohle, Madison, Wis.—p. 563.
Globulin Fraction in Rabbit's Plasma Possessing Strong Clotting Property. I. A. Parfentjev, Pearl River, N. Y.—p. 578.
Thrombic Activity of Globulin Fraction Derived from Rabbit Plasma. F. H. L. Taylor, E. L. Lozner and Margaret A. Adams, Boston.—p. 585.
*Use of Rabbit Thrombin as Local Hemostatic. E. L. Lozner, Harriet MacDonald, M. Finland and F. H. L. Taylor, Boston.—p. 593.

Comparison of Types of Insulin.—Duncan and Barnes state that there is evidence that globin insulin acts with sufficient rapidity to prevent hyperglycemia during the day and that its action is sufficiently prolonged to control the level of the blood sugar throughout the night. Before they attempted the routine treatment of diabetic patients with globin insulin they determined the time of onset, the intensity and the duration of the hypoglycemic effect produced in 42 patients by a single injection and compared the results with those produced by single doses of the insulins now in common use. The globin insulin, in all but 1 of the subjects studied, had a blood sugar lowering action which began within two hours of its injection. This effect reached a maximal intensity between the second and the eighth hour and was maintained for eighteen to twenty-four hours. The hypoglycemic action during the first four hours was slower than that of unmodified and crystalline insulin but more rapid than that of protamine zinc insulin. The period of maximal effect began sooner and ceased earlier than that following the administration of protamine zinc insulin. Thus the tendency of the more rapid action of globin insulin to prevent postcibal hyperglycemia would obviate the need of supplemental doses of unmodified or crystalline insulin. The lowest level of blood sugar was reached between the eighth and the tenth hour after the administration of globin insulin. Thus patients receiving globin insulin routinely are prone to have hypoglycemic reactions between 4 p. m. and dinner time unless appropriate dietary rearrangements are made to prevent them. Patients who received moderately large doses of insulin had slightly greater hypoglycemic effects from the globin than from the protamine zinc insulin, indicating that unit for unit globin insulin has a greater hypoglycemic effect than protamine zinc insulin.

Rabbit Thrombin as Local Hemostatic.—Lozner and his associates observed the effect of rabbit thrombin on 11 patients bleeding from small wounds. Nine of the patients had hemorrhagic diatheses (4 had hemophilia, 3 symptomatic thrombocytopenic purpura, 1 hypoprothrombinemia and 1 hereditary thrombasthenia) and 2 patients had hypertension with epistaxis. Dry, powdered rabbit thrombin was usually used to impregnate a dressing, pack or "gauze bite" dampened with sterile saline solution. The dry powder comes into immediate contact with the bleeding surface. With only one exception the hemostasis produced by rabbit thrombin was complete and immediate. That is, as soon as the dressing was applied hemorrhage was arrested. In 1 patient with hypertension and expistaxis bleeding had persisted for fourteen days prior to rabbit thrombin therapy, despite the use of various hemostatics and four blood transfusions. The patient in whom hemostasis was at first only partial had complete and immediate hemostasis after two subsequent applications on succeeding days. The unsatisfactory result of the first application was probably due to error in application. Rabbit thrombin solution was also of value as a local hemostatic but was not as effective as the dry powder. There were no toxic effects after the use of either form of thrombin. The hemostatic effect of rabbit thrombin is apparently accomplished by its instantaneous precipitation of fibrin.

American J. Obstetrics and Gynecology, St. Louis

42:557-744 (Oct.) 1941

Proliferative Changes in Senile Endometrium. E. Novak and E. H. Richardson Jr., Baltimore.—p. 564.
Comparative Analysis of Total Abdominal, Supravaginal and Vaginal Hysterectomies. H. E. Miller and O. Prejean, New Orleans.—p. 580.
Total Hysterectomy, Abdominal and Vaginal. W. C. Danforth, Evanston, Ill.—p. 587.
Excretion of Estrogen and Pregnandiol Preceding Normal Parturition. C. Bachman, Philadelphia.—p. 599.
Evaluation of Colorimetric Quantitation of 17-Ketosteroids: Application in Gynecology. R. A. Ross, E. C. Hamblen, W. K. Cuyler and Margaret Baptist, Durham, N. C.—p. 607.
Effect of Gonadotropins on Human Ovary. S. H. Geist, J. A. Gaines and U. J. Salmon, New York.—p. 619.
*Time of Onset and Duration of Toxemias of Late Pregnancy in Relation to Development of Permanent Vascular Damage. C. H. Peckham, Cooperstown, N. Y.—p. 638.
Interpretation of Blood Pressure Behavior During Pregnancy and Puerperium. R. A. Bartholomew and E. D. Colvin, Atlanta, Ga.—p. 646.
Endocrine Factors in Secondary Amenorrhea. C. F. Fluhmann and Kathleen M. Murphy, San Francisco.—p. 656.
Osteochondritis in Stillborn. J. R. McCord, Atlanta, Ga.—p. 667.
Study of Intrauterine Oxygen Exchange. N. R. Kretzschmar, H. A. Towsley, F. J. Stoddard and J. Engelfried, Ann Arbor, Mich.—p. 677.
Consideration of Cause and Possible Late Effect of Anoxia in Newborn Infant. F. L. McPhail and E. L. Hall, Great Falls, Mont.—p. 686.
*Vitamin Therapy in Vulvar Dermatoses: Components of B Complex. H. C. Hesseltine, Chicago.—p. 702.
Developments and Trends in Gynecology. H. E. Sigerist, Baltimore.—p. 714.

Pregnancy Toxemias in Relation to Vascular Damage.—Peckham followed up 500 women whose pregnancy was complicated by toxemia to determine whether the time of onset and the duration before parturition are of importance in the development of permanent vascular damage. The follow-up study after parturition consisted in examinations at intervals of three months for two to twelve years. All the women were apparently normal when first seen in the antepartum clinic. The race and age of the patient, the degree of hypertension, the degree of albuminuria during pregnancy, the type of delivery and the result to the child had no significant bearing on the month in which toxemia developed. The incidence of multiparas increased as the onset of the toxemia took place in the earlier months of gestation. If the toxemia was manifest near the beginning of gestation the number of abnormally high blood pressure readings increased, both at the time of the patient's discharge from the hospital and six weeks post partum. The incidence of previous toxemia was higher in patients evidencing toxemia early in pregnancy than in those whose first abnormality occurred in the later months. There was a similar trend in subsequent pregnancies. The incidence of chronic vascular damage was 63.4 per cent. The percentage was 89.93 among multiparas and 32.76 among primiparas. Vascular damage was always seen in the primipara whose toxemia occurred prior to the sixth month of gestation. Multiparas had vascular damage if the toxemia occurred prior to the eighth month. Vascular damage

occurred in the primipara who had been carried for more than twelve weeks after toxemia developed; the limit for multiparas was six weeks. It seems that the duration of a toxemia is of more importance in relation to subsequent vascular disease than the month of onset. Termination of the pregnancy to avoid chronic damage appears justified if signs and symptoms persist after four weeks of observation and treatment.

Vitamin Therapy in Vulvar Dermatoses.—Hesseltine observed the responses of 15 patients with chronic atrophic dermatitis of the vulva, 5 with an undetermined type of vulvitis and 2 with unexplained vulvar pruritus to single and combined factors of the vitamin B complex. The oral route was used almost exclusively. According to the usual standards, 19 of the 22 patients were apparently free of vitamin deficiency, 1 existed on an inadequate diet, 1 had a faulty or inadequate absorption and 1 suffered from a protracted and unusual infection with multiple draining areas. If improvement did not follow thiamine hydrochloride therapy other components of the B complex were added. Therapy with the vitamin B complex did not eradicate vitiligo or correct advanced chronic atrophic dermatitis of the vulva, although symptoms were partially or completely relieved and fissures and leukoplakic areas disappeared. On the other hand the first stage of chronic atrophic vulvitis and certain types of undetermined vulvitis cleared up after treatment with thiamine hydrochloride, thiamine hydrochloride and riboflavin or multiple factors of the B complex. There was a tendency to recurrence in a few of the patients whose symptoms were alleviated (cured), possibly because of the premature withdrawal of the vitamins, some imbalance in metabolism or some other state. Small maintenance doses may be needed. The administration of factors of the B complex for chronic atrophic dermatitis of the vulva is not yet endorsed except for persons in such poor health they cannot be operated on and for patients with a vulvitis for which there is no specific therapy. If chronic irritation can be relieved perhaps the incidence of malignant growths might be reduced.

American Journal of Psychiatry, New York

98:159-316 (Sept.) 1941

Usefulness of Statistics to the Administrator. N. A. Dayton, Boston.—p. 159.

One Hundred Schizophrenic Cases of Late Hospitalization. L. Maletz and Grace H. Kent, Hathorne, Mass.—p. 173.

Comparison of Results of Metrazol Therapy with Group of Matched Controlled Cases. J. B. Craig and M. E. Schilling, Ann Arbor, Mich.—p. 180.

Electroencephalogram Studies of Nine Cases with Major Psychoses Receiving Metrazol. K. H. Finley and J. M. Lesko, Boston.—p. 185.

Protracted Shock: Its Cause and Its Prevention. J. P. Frostig, San Francisco; I. M. Rossman, W. B. Cline Jr. and O. Schwoerer, Wingdale, N. Y.—p. 192.

Effect of Benzedrine Sulfate Alternated with Sodium Amytal in Schizophrenia. L. Reznikoff, Secaucus, N. J.—p. 196.

Shock Therapy in Psychosis Complicating Pregnancy: Case Report. H. H. Goldstein, J. Weinberg and M. I. Sankstone, Chicago.—p. 201.

General Semantics, Psychiatry, Psychotherapy and Prevention. A. Korzybski, Chicago.—p. 203.

*Amphetamine (Benzedrine) Sulfate as Corrective for Depression of Sedative Medication in Epilepsy. L. J. Robinson, Palmer, Mass.—p. 215.

Physiologic Conception and Treatment of Certain Common "Psychoneuroses." E. Jacobson, Chicago.—p. 219.

X-Ray Evidence of Emotional Influences on Esophageal Function. W. B. Faulkner Jr., San Francisco.—p. 227.

Psychopathic Department of an American General Hospital in 1808. W. L. Russell, New York.—p. 229.

Method for Preliminary Psychiatric "Screening" of Large Groups. L. S. Penrose, London, Ont., Canada, and C. R. Myers, Toronto, Canada.—p. 238.

Disturbances of Behavior in Patients with Disseminated Sclerosis. O. R. Langworthy, L. C. Kolb, Baltimore, and S. Androp, Catonsville, Md.—p. 243.

Electroencephalogram of Normal Children: Effect of Hyperventilation. N. Q. Brill and Herta Seidemann, New York.—p. 250.

Amphetamine Sulfate as Corrective in Epilepsy.—Robinson gave amphetamine sulfate to 58 epileptic patients to circumvent the untoward toxic effects (irritability, drowsiness, ataxia, aggressiveness, anorexia, vertigo, nystagmus and tremor) of anticonvulsant therapy. The therapy usually consisted of phenobarbital. The daily dose of amphetamine sulfate ranged from 5 to 30 mg. It was given orally. The drug counteracted the drowsiness in 39, or 64.2 per cent, of the 56 patients having this manifestation. It combated along with the drowsiness the ataxia in 3 of 13 patients. It counteracted irritability in 3 of 15 patients. Giving amphetamine sulfate did not usually correct aggressiveness. It was necessary to give amphetamine sulfate only temporarily to patients who slowly acquire a tolerance to large doses of phenobarbital. In such patients it overcame the toxic symptoms until tolerance was established, when it could be withdrawn. In other patients, toxic phenomena reappeared unless the amphetamine sulfate therapy was continued as long as the phenobarbital medication was not reduced. In no case did the use of amphetamine sulfate alter the incidence of seizures. What it did do was to make it possible to reduce seizures in many patients by giving them adequate anticonvulsant medication without toxic manifestations forcing discontinuance.

Archives of Dermatology and Syphilology, Chicago

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Balanitis Xerotica Obliterans. C. Freeman, St. Paul, and C. W. Laymon, Minneapolis.—p. 547.

Membranous Stomatitis Associated with Debilitation and Uremia. E. S. Bereston, Baltimore, and H. Keil, New York.—p. 562.

Photodynamic Action of Lime Oil (Citrus Aurantifolia). W. M. Sams, Miami, Fla.—p. 571.

Argyria, with Special Reference to Cutaneous Histopathology. W. R. Hill and H. Montgomery, Rochester, Minn.—p. 588.

Coral Dermatitis. O. L. Levin and H. T. Behrman, New York.—p. 600.

Dermatitis Venenata Due to Nail Lacquer. E. D. Osborne, J. W. Jordon and P. C. Campbell Jr., Buffalo.—p. 604.

Erythema Palmare and Naevus-Araneus-like Telangiectases. E. N. Walsh, Whiting, Ind., and S. W. Becker, Chicago.—p. 616.

Combined Fungous Infections: Report of Six Cases with Review of Thirty-Six Cases from Literature. E. Muskatblit, New York.—p. 631.

Histologic Studies of Uninvolved Skin of Patients with Psoriasis. J. F. Madden, St. Paul.—p. 655.

Solubility of Dermatitis-Producing Fraction of Poison Ivy. J. B. Howell, Dallas, Texas.—p. 665.

Syphilis of Larynx: Report of Atypical Case. L. Felderman, Philadelphia.—p. 667.

Urticaria Due to Tryparsamide. R. H. Kampmeier, Nashville, Tenn.—p. 671.

44:773-982 (Nov.) 1941

Ectodermal and Mesodermal Dysplasia with Osseous Involvement. H. N. Cole, J. R. Driver, H. K. Giffen, C. B. Norris and G. Stroud Jd, Cleveland.—p. 773.

Acanthosis Nigricans: Its Occurrence in Association with Gastric Carcinoma in Seventeen Year Old Girl. W. C. Herold, St. Louis; W. H. Kaufman and D. C. Smith, Charlottesville, Va.—p. 789.

*Iontophoresis of Copper Sulfate in Cases of Proved Mycotic Infection. A. M. Greenwood and Ethel M. Rockwood, Boston.—p. 800.

Undenatured Trichophytin: Preparation and Clinical Application. H. E. Miller, R. L. Sayers and Frances Kimura, San Francisco.—p. 804.

Trichophytin: II. Apparent Separation of Skin-Reactive Factor from Therapeutic Principle in Trichophytin. S. M. Peck, A. Glick and E. Weissbard, New York.—p. 816.

*Ringworm of Feet: Shoes and Slippers as Source of Reinfection: Final Report. R. C. Jamieson and Adelia McCrea, Detroit.—p. 837.

Ingestion of Lard in Treatment of Eczema and Allied Dermatoses: Clinical and Biochemical Study. C. W. Finnerud, Chicago; R. L. Kesler, Oak Park, Ill., and Hilda F. Wiese, Chicago.—p. 849.

Absorption of Externally Applied Ammoniated Mercury. O. S. Gibbs, R. Shank, Harriet Pond, Memphis, Tenn., and G. H. Hansmann, Milwaukee.—p. 862.

*Sulfaguanidine in Treatment of Proctitis Due to Lymphogranuloma Venereum: Report of Six Cases. O. Cañizares, New York, and G. E. Morris, Boston.—p. 873.

Accidental Inoculation with Spirocharta Pallida. C. Shaw, Chattanooga, Tenn.—p. 878.

Histamine in Treatment of Atopic Dermatoses. L. M. Smith, El Paso, Texas.—p. 883.

Localized Amyloidosis of Skin: Report of Cases; Intracutaneous Congo Red Test as Diagnostic Aid. A. Dostrovsky and F. Sagher, Jerusalem, Palestine.—p. 891.

Postular Psoriasis in Negress. H. Nieman, Dayton, Ohio.—p. 907.

Iontophoresis of Copper Sulfate for Mycotic Infection.—Greenwood and Rockwood say that for their 4 patients from whom material which produced cultures of identifiable fungous organisms was obtained from affected areas at their first visit the introduction of copper sulfate into the skin by iontophoresis was neither fungicidal nor fungistatic for the three pathogens found: Trichophyton gypseum, Trichophyton purpureum and Epidermophyton inguinale. When clinical improvement follows such treatment it must be due to some factor or factors other than the copper sulfate. One of the patients was given twenty-five treatments, and still it was possible to grow T. gypseum from scales taken at each examination and at the patient's last visit. In another patient the process was extending so rapidly after the sixth iontophoresis that compound ointment of benzoic acid was applied for ten days, and since then there

has been no obvious activity and all scales and cultures have been negative for T. gypseum. The third patient was treated for a month, and culture for E. inguinale was positive after each treatment. After treatment with compound ointment of benzoic acid the condition was clinically improved but still active mycologically for E. inguinale. The fourth patient was treated with copper sulfate iontophoresis for thirty-three days, and culture of material obtained during and at the conclusion of treatment showed T. purpureum.

Ringworm of Feet.—Jamieson and McCrea found known organisms in scrapings from the shoes of 40 per cent of patients with ringworm of the feet; the specimens from 25 per cent were definitely not infected, and those from 34 per cent were doubtfully infected. The organisms found in the shoes varied in number and type according to the length of time the shoe had been worn, the severity of the infection, the care and protection given the feet and the frequency with which stockings were changed. A study of whether the pathogen found in the scrapings from the skin and that in scrapings from the soles of the shoes were the same revealed accepted pathogens (Trichophyton, Microsporon and Epidermophyton) in the skin of 18 of 25 patients but in the scrapings from the shoes of only 12. The scrapings from the shoes of these 12 showed the same organisms found in cultures of scrapings from the skin. The number of possible pathogenic fungi (Monilia, Actinomyces, Torula, yeast and Penicillium brevicaule) found in scrapings from the skin and from shoes was nearly equal, twenty and twenty-one respectively. In addition to the accepted and possible pathogens, various accepted nonpathogenic organisms were revealed in fourteen cultures of scrapings from the skin and in thirty-one cultures of scrapings from shoes. This result seems natural if the lack of care the shoe receives compared with that given the foot is considered.

Sulfaguanidine for Proctitis.—Cañizares and Morris used sulfaguanidine for the treatment of 6 patients with proctitis due to venereal lymphogranuloma. The daily dose of the drug was 24 Gm., divided into four equal doses. The period of treatment varied from twenty-seven to forty-seven days. All patients with simple inflammatory proctitis experienced subjective and objective improvement. Patients with narrow fibrotic stricture of the rectum were not improved; to them the drug was given preoperatively as a preparatory measure. Chemotherapy will not do more than heal erosions and ulcerations.

Archives of Neurology and Psychiatry, Chicago

46:761-946 (Nov.) 1941

Vascular Supply of Spinal Ganglions. L. Bergmann, Boston, and L. Alexander, Durham, N. C.—p. 761.
Pathology of Amyotrophic Lateral Sclerosis: Fiber Analysis of Ventral Roots and Pyramidal Tracts of Spinal Cord. G. Wohlfart and R. L. Swank, Boston.—p. 783.
Studies in Diseases of Muscle: X. Prostigmine and Physostigmine in Treatment of Myasthenia Gravis. A. T. Milhorat, with technical assistance of W. E. Bartels, New York.—p. 800.
Neurologic Symptoms Following Extensive Occlusion of Common or Internal Carotid Artery. A. B. King and O. R. Langworthy, Baltimore.—p. 835.
*New Method for Treatment of Cystic Craniopharyngioma by Intraventricular Drainage. J. E. Scarff, New York.—p. 843.
Roentgenologic Changes in Bones in Cases of Pseudohypertrophic Muscular Dystrophy. B. S. Epstein and J. L. Abramson, Brooklyn.—p. 868.
Topical Arrangement Within Spinothalamic Tract of Monkey. T. A. Weaver Jr., Cincinnati, and A. E. Walker, Chicago.—p. 877.
Metrazol Convulsions in Treatment of Psychosis of Dementia Paralytica. Vivian Bishop Kenyon, Osawatomie, Kan.; M. Lozoff and D. Rapaport, Topeka, Kan.—p. 884.
*Dilantin Hyperplastic Gingivitis: Its Treatment and Prevention. D. E. Ziskin, L. R. Stowe and E. V. Zegarelli, New York.—p. 897.
Removal of Tumor Arising Anterior to Medulla. A. Ecker, Syracuse, N. Y.—p. 908.

Intraventricular Drainage for Cystic Craniopharyngioma.—Scarff treated 7 patients with suprasellar cysts by internal drainage into the cerebral ventricles. The basic principle of the new method is that it establishes a communication between the dome of the cyst and the floor of the lateral ventricle. Such an opening, if adequately made, will probably remain patent, allowing permanent drainage of the secretions issuing from the lining of the cyst into the cerebrospinal fluid, which carries away the secretions in its regular circulation. In selected instances (with a cystic craniopharyngioma large enough to push up and thin out the floor of the lateral ventricles) the method promises better results than those that have been obtained by draining the contents of the cyst.

Phenytoin Hyperplastic Gingivitis.—Ziskin and his associates studied the gums of 14 epileptic patients, aged from 9 to 44 years, taking phenytoin sodium daily. Three boys, aged 8, 13 and 17 years, respectively, whose seizures were controlled with phenobarbital, were included for comparison. The chief complaints of the patients receiving phenytoin sodium were bleeding from the gums, enlargement of the gums and difficulty in mastication. Without therapy, phenytoin gingivitis progresses steadily. As proliferation progresses, the teeth are moved out of their natural alinement, and the interseptal bone may be resorbed, producing looseness and sometimes loss of the teeth. If the hyperplastic tissue is extracted the appearance of the gums returns to normal for a time without further treatment. Vigorous massage of the interdental papillae or surgical intervention followed by massage was more successful. Gingival treatment should probably be begun when phenytoin therapy is started. Administration of the drug need not be discontinued because of the gingival hyperplasia. Other sedatives used in the control of epileptic seizures do not stimulate the gums to hyperplasia. The resemblance of the phenytoin gingival hyperplasia to the hypertrophy seen in scurvy is superficial; the hypertrophy in scurvy is caused by swelling and not by hyperplasia as it is in phenytoin gingivitis. Pain, marginal necrosis of the gums and occasional petechial hemorrhage are absent in phenytoin hyperplasia, as are constitutional purpura and soreness of the joints.

Delaware State Medical Journal, Wilmington

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Psychosomatic Medicine and the General Physician. E. Weiss, Philadelphia.—p. 181.
Osteomalacia. H. G. Hadley, Washington, D. C.—p. 184.

Diseases of Eye, Ear, Nose and Throat, Chicago

1:289-324 (Oct.) 1941

Present Status of Roentgen Therapy in Otologic Disease. R. Schillinger, New York.—p. 294.
Visual Psychology in War Time. P. Fridenberg, New York.—p. 298.
*Ethmosphenoidal Epiglottidean Syndrome Interfering with Voice Dynamics. L. Felderman, Philadelphia.—p. 300.
Blunt Injuries to Eye and Adnexa. E. E. Grossmann, Camp Hulen, Texas.—p. 309.
Deep Infections in Neck. S. Iglauer, Cincinnati.—p. 313.

Ethmosphenoidal Epiglottidean Syndrome and Voice Dynamics.—Felderman describes an impairment of the vocal mechanism, ethmosphenoidal epiglottidean syndrome, that he encountered over a decade in 67 patients. He believes the syndrome to be more frequent than his observations indicate. The patients complain, usually in the morning, that they are not in "good voice." The alteration in voice and the hoarseness clear up during the day. Vocalization and swallowing usually clear up the effete mass that has accumulated during the night. The patient finds it difficult to start phonation, and his throat tires quickly. Pus may be found in the nasopharynx. Partial or total loss of taste or smell, a ticklish and tight feeling about the throat and blocking of the ears, with or without spasmodic cough, are some of the symptoms. Indirect pressure on Jacobson's nerve may give rise to otic symptoms. Patients with the syndrome also show a faint redness of the tympanic membrane, with or without a corresponding impairment in hearing. Less frequently they hear tingling bells, an echo, a vibration or the reechoing of a note in lesser or greater intensity. Another group of symptoms, mostly pharyngeal, arises when the muscles of the pharynx and its nerve supply are involved. If pressure on the laryngeal structure from even a trivial amount of pus is present it is sufficient to interfere with the normal emission of sounds. In the beginning the patient may rid himself of the pus by coughing. Later pus accumulates in larger quantities and remains in evidence when the epiglottis is examined. The accumulated pus pressing on the vallecula of the epiglottis is sufficient to upset the normal working balance of the intrinsic group of muscles. To a singer, the "head cold," if not treated properly, is one of the most devastating of human maladies, as it may leave an infective focus in the ethmosphenoidal area.

The syndrome has been mistakenly diagnosed as pachydermia of the larynx, prolapse of the laryngeal ventricle, singers' nodules, hemorrhage of the vocal cords, arthritis, perichondritis of the larynx, thyroid disorders, syphilis and as early tuberculosis and cancer of the larynx. It takes eight to ten weeks for the voice to become normal again after surgical or nonsurgical treatment. The downward gravitation of the infection into the many inaccessible and complex structures makes it difficult to eradicate. When a mechanical obstruction exists the most essential and conservative surgical intervention is indicated. Infected tonsils should be removed and dental caries treated. In formulating a plan of nonsurgical treatment, one should correct any endocrine and vitamin deficiencies and allergic tendencies. Autogenous vaccine has been helpful. A mild germicide (0.2 or 0.1 of 1 per cent silver picrate in 7.5 per cent dextrose solution) will diminish the production of pus without paralyzing the ciliary activity of the nose.

Endocrinology, Springfield, Ill.

29:291-482 (Sept.) 1941. Partial Index

Effect of Pituitary Growth Substance on Development of Rats Thyroidectomized at Birth. Theodora Nussmann Salmon, New York.—p. 291.

Profound Vascular Changes Induced in Uterus of Castrated Rabbit by Combinations of Estradiol Benzoate and Progesterone. J. Gillman, Johannesburg, South Africa.—p. 336.

Effect of Acute Estradiol Overdosage on Uterus of Guinea Pig. C. Dosne, Montreal, Canada.—p. 352.

Comparative Estrogenic Potency of Diethylstilbestrol, Estrone, Estradiol and Estriol: II. Uterine and Vaginal Changes in Infantile Rats. J. L. Sealey and C. W. Sondern, Kansas City, Mo.—p. 356.

Comparative Studies of Effects of Estradiol and Stilbestrol on Blood, Liver and Bone Marrow. D. Castrodale, Olga Bierbaum, E. B. Helwig and C. M. Macbryde, St. Louis.—p. 363.

Effect of Estrogen on Permeability of Uterine Capillaries. O. Hechter, L. Krohn and J. Harris, Los Angeles.—p. 386.

Effect of Testosterone Propionate on Mitotic Activity of Adrenals in Intact Immature Female Rat. I. T. Nathanson and A. M. Brues, Boston.—p. 397.

Forty-Eight Hour Response of Immature Male Rat to Androgens. R. R. Greene and M. W. Burrill, Chicago.—p. 402.

Fractionation of Neutral Urinary Steroids. G. Pincus and W. H. Pearlman, Worcester, Mass.—p. 413.

Influence of Adrenal Preparations on Basal Metabolism and Specific Dynamic Action. Katharine A. Brownell and F. A. Hartman, Columbus, Ohio.—p. 430.

Reversal of Fatigue in Adrenalectomized Rat by Glucose and Other Agents. D. J. Ingle and F. D. W. Lukens, Philadelphia.—p. 443.

Action of Noxuous Agents on Insulin and Adrenalin Sensitivity, and on Glucose Tolerance. G. Masson, Montreal, Canada.—p. 453.

Some Effects of Constant Intravenous Injection of Potassium Chloride Solutions in Normal Conscious Dogs. H. M. Schamp, Chicago.—p. 459.

Influence of Thyroid on Vagoinsulin and Sympatheticoadrenal Systems. E. Gellhorn and J. Feldman, Chicago.—p. 467.

Journal of Allergy, St. Louis

12:523-638 (Sept.) 1941

Active Anaphylaxis in Chick Embryo: Preliminary Report. F. W. Wittich, Minneapolis.—p. 523.

Use of Sulfonamide Drugs in Bronchial Asthma: Preliminary Report of Twenty-Three Cases. L. Unger, Chicago.—p. 528.

Use of Bovine Antitoxin for Prophylaxis of Tetanus. J. Glaser, Rochester, N. Y.—p. 537.

Immunologic Studies with Conjugated Ragweed Pollen Extracts. Margaret B. Strauss and W. C. Spain, New York.—p. 543.

Studies on Blocking Antibody of Cooke in Treatment of Hay Fever. Margaret A. Scully and F. M. Rackemann, Boston.—p. 549.

Abortion After Grass Pollen Injection. N. Francis, Rochester, N. Y.—p. 559.

Studies in Epinephrine: Recovery of Epinephrine Injected Intravenously and Subcutaneously. S. S. Bullen and W. R. Bloor, Rochester, N. Y.—p. 564.

Pollen Studies: Contrast Color Method of Examining Unstained Pollen Grains. R. I. Alford, New York.—p. 572.

Henoch's Purpura Based on Food Allergy: Report of Two Cases. S. F. Hampton, St. Louis.—p. 579.

A 1:500 Epinephrine in Gelatin: Discussion of Its Action, Advantages and Disadvantages. S. D. Lockey, Lancaster, Pa.—p. 592.

Psyllium Seed Sensitivity: Case Report. M. S. Ascher, Detroit.—p. 607.

Allergic Skin Reactions to Mammalian Serums. F. A. Simon, Louisville, Ky.—p. 610.

Absorption of Injected Pollen Antigens: Comparison of Subcutaneous and Intramuscular Routes. S. J. Levin and L. E. Heideman, Detroit.—p. 616.

Journal of Clin. Endocrinology, Springfield, Ill.

1:633-710 (Aug.) 1941

Testosterone Therapy of Eunuchoids: II. Clinical Comparison of Parenteral Implantation and Oral Administration of Testosterone Compounds in Male Eunuchoidism. R. F. Escamilla and H. Lisser, San Francisco.—p. 633.

Stilbestrol Induced Testicular Degeneration in Hypersexual Males. C. W. Dunn, Philadelphia.—p. 643.

Results of Treatment in Forty Selected Cases of Cryptorchism. N. H. Einhorn and L. G. Rountree, Philadelphia.—p. 649.

Methyl Testosterone: IV. Observations on Hypermetabolism Induced by Methyl Testosterone. R. Jones, E. P. McCullagh, D. R. McCullagh and G. W. Buckaloo, Cleveland.—p. 656.

Effect of Female Sex Hormone on Volume of Seminal Fluid in Man. N. J. Heckel and C. R. Steinmetz, Chicago.—p. 664.

Colorimetric Determination of Neutral Steroids (Hormones) in Twenty-Four Hour Sample of Human Urine (Pregnandiol; Total, Alpha and Beta Alcoholic and Nonalcoholic 17-Ketosteroids). N. B. Talbot, R. A. Berman, E. A. MacLachlan and J. K. Wolfe, Boston.—p. 668.

Prophylactic Implantation of Estrogens in Ovariectomized Women. U. J. Salmon, S. H. Geist and R. I. Walter, New York.—p. 674.

Effective Therapy of Menopause Using Estrogen in Lower Dosage. P. M. Jolfe, Paterson, N. J.—p. 677.

Pitressin Tannate Therapy in Diabetes Insipidus. G. W. Thorn and Kay E. Stein, Baltimore.—p. 680.

Simmonds' Disease with Craniopharyngioma. J. E. Farber, K. Goldstein and W. F. Beswick, Buffalo.—p. 688.

Contributions from United States of America to Endocrinology. E. L. Sevringhaus, Madison, Wis.—p. 691.

Journal Industrial Hygiene & Toxicology, Baltimore

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Compensation of Occupational Diseases. L. Teleky, New York.—p. 353.

*Industrial Manganese Poisoning. R. H. Flinn, P. A. Neal, Bethesda, Md., and W. B. Fulton, Harrisburg, Pa.—p. 374.

Effects of Carbon Disulfide on Blood Corpuscles. H. Brieger, Philadelphia.—p. 388.

Dropping Mercury Electrode for Lead Analysis. E. C. Barnes and H. W. Speicher, East Pittsburgh, Pa.—p. 397.

Chemical Changes of Methyl Bromide in Animal Body in Relation to Its Physiologic Effects. D. D. Irish, E. M. Adams, H. C. Spencer and V. K. Rowe, Midland, Mich.—p. 408.

Industrial Manganese Poisoning.—According to Flinn and his co-workers, 11 of 34 men who had worked or were working in an atmosphere containing manganese had varying degrees of chronic manganese poisoning. Ten of the 11 men complained of lassitude, drowsiness, tremor of the body or the extremities, disturbances of gait and slight dyspnea; 9 of muscular weakness and muscular cramps; 8 of disturbances in speech and sexual function; 7 of metallic taste; 6 of vertigo, paresthesia and palpitation; 5 of loss of appetite, muscular pain, twitching of fingers and headache; 4 of articular pains and increased perspiration; 3 of salivation, impulsive weeping and laughter, and 2 of difficulty in swallowing, violent temper and nausea. A low leukocyte count with a reduced percentage of neutrophilic leukocytes was a significant finding, but the authors do not know whether or not this occurs early enough to be used as a criterion for removing a worker from exposure to manganese. As with other occupational diseases, the first step in the diagnosis is to obtain the occupational history and knowledge of any diseases associated with such a history. From the study of the course of the 11 patients it appears that many of the symptoms will disappear or regress if the worker is removed from contact with manganese shortly after the onset of symptoms. Some residual disturbances in gait and speech may remain. Well advanced manganese poisoning is a permanent crippling disease, particularly of the legs. Many of the symptoms will improve slowly, with occasional remissions, but the use of the legs is only partially recovered. Quarterly physical examinations should be made of all employees who work in an atmosphere of manganese dust, and any workers showing signs of early poisoning (drowsiness, languor, muscular cramps, twitching or tremor) should be transferred to an environment free of manganese until the hazard has been controlled. Later symptoms and signs (disturbances in gait and speech) may mean that the disease has progressed too far to be relieved by removing the subject from exposure to manganese. Engineering control methods in general use in dusty trades are effective in preventing the dispersal of manganese dusts.

Journal of Lab. and Clinical Medicine, St. Louis

26:1867-2014 (Sept.) 1941

Allergy of Abdominal Organs. M. Walzer, Brooklyn.—p. 1867.
Familial Nonreaginic Food Allergy: Its Specific Diagnosis and Treatment. A. F. Coca, Oradell, N. J.—p. 1878.
Left Ventricular Failure Due to Essential Hypertension. N. Flaxman, Chicago.—p. 1891.
Acute Edematous Pancreatitis: Case Presentation and Clinical Discussion of Serum Amylase Test. E. F. Lewison, Baltimore.—p. 1901.
High Incidence of Infective Stools in Small Outbreak of Infantile Paralysis. G. Y. McClure, Albany, N. Y.—p. 1906.
Behavior of Recipient's Leukocyte Count Following Transfusion of Preserved Blood. J. A. Graham and W. M. Fowler, Iowa City.—p. 1911.
Effects of Decreased Temperature on Activity of Intact Muscle. W. W. Tuttle, Iowa City.—p. 1913.
Study of Therapeutic Effect of Sulfapyridine in Pneumococcus Infected Mice in Atmospheres of Varying Oxygen Tension. A. L. Barach and N. Molomut, New York.—p. 1915.
Gold Colloid and Colloids of Other Heavy Metals in Treatment of Rheumatoid Arthritis. J. M. Tarsy, Brooklyn.—p. 1918.
Relation of Nasopharynx to Ulcerative Colitis: Preliminary Report. S. Weiss, A. Slanger and S. Goodfriend, New York.—p. 1925.
Effects of Insulin, Metrazol and Electric Shock on Blood Pyruvate, Lactate and Glucose. K. A. C. Elliott, T. D. Rivers, F. H. Elliott and B. Platt, Philadelphia.—p. 1928.
Blood Specific Gravity Studies: Relationship of Specific Gravity of Whole Blood to Specific Gravity of Plasma, Red Blood Cell Count, Hematocrit and Hemoglobin as Indicators of Hemoconcentration. C. T. Ashworth and G. Adams, Dallas, Texas.—p. 1934.
Indoluria in Rheumatoid Arthritis. E. Neuwirth, New York.—p. 1939.
Iodine Content of Blood, Urine and Saliva of Normal Persons in New York City Area. M. Bruger, J. W. Hinton and W. G. Lough, New York.—p. 1942.

27:1-130 (Oct.) 1941

Experimental Study of Lymphocytic Response in Thyrotoxicosis. L. A. Turley and K. M. Richter, Oklahoma City.—p. 1.
Statistical Study of Temperatures in Hypoglycemic Coma. Esther Bogen Tietz, Cecile Nadler and Edith Klemperer, Cincinnati.—p. 11.
Serologic Diagnosis in Histologically Proved Chronic Syphilis: Analysis of 424 Necropsies. O. A. Brines and B. Juliar, Detroit.—p. 15.
Diagnosis of Peripheral Arterial Obstruction. S. S. Samuels, New York.—p. 19.
Sensitization Induced by Tetanus Toxoid, Alum Precipitated. H. Gold, Chester, Pa.—p. 26.
Localization and Concentration of Staphylococcus Antitoxin in Areas of Rabbit's Skin. R. H. Rigdon, Memphis, Tenn.—p. 37.
*Bacteremia Produced by Aerobic, Gram-Negative Sporulating Bacillus. A. Bondi Jr., E. H. Spaulding and J. A. Kolmer, Philadelphia.—p. 41.
Separation of Coagulant from Toxic Principles of Venom of Australian Tiger Snake (Notechis Scutatus), with Remarks on Mode of Action of Coagulant. S. Rosenfeld and J. Rubinstein, Brooklyn.—p. 45.
Studies on Detection of Abscesses and Tumors: III. Concentration and Detection of Radioactive Substance in Abscesses. H. H. Kroll, S. F. Strauss and H. Nechelis, Chicago.—p. 50.
Studies in Absorption of Undigested Protein: IX. Absorption from Stomach and Esophagus. M. Harten, I. Gray, S. Livingston and M. Walzer, Brooklyn.—p. 54.
Identification of Blastomycoides Histolytica in Three Infections of Central Nervous System. Rebecca A. Holt, Richmond, Va.—p. 58.
*Chemical Studies in Delirium Tremens. C. Cohn, Philadelphia.—p. 63.
Effect of Intravenous Administration of Phosphate Solution in Normal Rabbits. I. Greenfield, Brooklyn.—p. 68.

Bacteremia Produced by Aerobic Bacillus.—Bondi and his associates isolated an aerobic, gram-negative, spore-forming bacillus from the blood of 5 hospitalized patients. The bacillus had no serologic relationship to Alcaligenes fecalis. Over a period of five months the bacillus was isolated nine times: once from each of 3 patients, twice from 1 and four times from 1 patient. It was isolated also from the bone marrow and from the spleen at the necropsy of the last patient nine days after the last blood culture was made. The strains in all 5 patients were identical; evidently they comprised a single species. The consistency with which this bacillus was isolated in cultures of blood from the 1 patient convinced the authors that they were not dealing with an ordinary contaminant but with an actual inhabitant of the blood stream. They describe this bacillus and report the results of serologic and pathogenic studies.

Chemical Studies in Delirium Tremens.—Cohn studied the blood constituents of 15 male patients with delirium tremens on admission and after recovery. Treatment varied, but generally it consisted in giving sodium chloride, fluids, thiamine hydrochloride and nicotinic acid orally or parenterally. On admission the serum chloride and carbon dioxide combining power were below normal in nearly all patients. Determinations of total base and available fluid showed low or low normal values. Hemoglobin and serum protein concentrations and the hematocrit values were increased. In the spinal fluid the amount of chloride was decreased and that of protein increased. On recovery of the patient the concentrations of the substances studied returned to normal, with the occasional exception of the concentration of the serum chloride. All but 1 patient recovered; this patient died within twenty-four hours of admission from lobar pneumonia. Recovery was accompanied by a restoration of body fluids and electrolytes to normal composition. Delirium tremens should be treated by methods which restore fluid and salt to the body.

Journal of Nutrition, Philadelphia

22:333-438 (Oct.) 1941. Partial Index

Influence of Plane of Nutrition and of Environmental Temperature on Relationship Between Basal Metabolism and Endogenous Nitrogen Metabolism Subsequently Determined. R. Treichler and H. H. Mitchell, Urbana, Ill.—p. 333.
Pathology of Riboflavin Deficiency in the Rat. J. H. Shaw and P. H. Phillips, Madison, Wis.—p. 345.
Vitamin B Complex and Fat Metabolism. J. C. Forbes, Richmond, Va.—p. 359.
Observations on Induced Caries in Rats: III. Effect of Fluoride on Rat Caries and on Composition of Rats' Teeth. F. J. McClure, Bethesda, Md.—p. 391.
Studies on Rat Growth Assay Method for Riboflavin. H. R. Street, New York.—p. 399.
Riboflavin Content of Fish Products. F. L. Billings, J. Biely, H. Fisher and C. Hedrees, Vancouver, B. C., Canada.—p. 425.
Effect of Autoclaving on Nutritive Value of Proteins in Cottonseed Meal. H. S. Olcott, Albany, N. Y., and T. D. Fontaine, New Orleans.—p. 431.

Journal of Pediatrics, St. Louis

19:289-436 (Sept.) 1941

General Considerations: Certain Problems of Puberty and Adolescence. L. K. Frank, New York.—p. 294.
Some Observations on Growth and Development of Adolescent Children. W. W. Greulich, Cleveland.—p. 302.
Examination of the Adolescent Female. J. L. Baer, Chicago.—p. 315.
Menstrual Abnormalities of Adolescence. E. L. Sevringhaus, Madison, Wis.—p. 319.
Endocrine Problems in Adolescence. E. Shorr, New York.—p. 327.
Standard Metabolism of Adolescence. B. Webster, Helen Harrington and L. M. Wright, New York.—p. 347.
Obesity in Relation to Puberty. Hilde Bruch, New York.—p. 365.
What to Do About the Fat Child at Puberty. F. W. Schlutz, Chicago.—p. 376.
Athletic Activity at Puberty, with Special Reference to the Cardiac and Tuberculous Patient. S. Gibson, Chicago.—p. 382.
Sexual Education of the Adolescent. G. J. Mohr, Chicago.—p. 387.
Psychologic Aspects of Adolescence. D. A. Thom, Boston.—p. 392.
Use of Bovine Antitoxin for Prophylaxis of Tetanus. J. Glaser, Rochester, N. Y.—p. 403.

19:437-578 (Oct.) 1941

Physiology and Anatomy of Respiratory System in Fetus and Newborn Infant. W. F. Windle, Chicago.—p. 437.
Neurotropic Virus Infections of Man. A. B. Sabin, Cincinnati.—p. 445.
Congenital Heart Disease and Electrocardiogram. G. Eisenberg and S. Gibson, Chicago.—p. 452.
Clinical Study Concerning Value of Biolac: I. As Suitable Food Substance: II. As Source of Available Iron for Infants. R. C. Eley, Boston.—p. 470.
*Cutaneous Reactions with Specific Soluble Substance in Infants and Children. R. Meyer and B. W. Carey, Detroit.—p. 481.
*Effect of Honey on Calcium Retention in Infants. E. M. Knott, C. F. Shukers and F. W. Schlutz, Chicago.—p. 485.
Influence of Diet of Newborn Infant on Prothrombin Index. S. S. Gellis and R. A. Lyon, Cincinnati.—p. 495.
Prothrombin in Newborn Infant: IV. Further Observations on Prothrombin Response to Intravenous Administration of Water Soluble Naphthoquinone. S. Kove and H. Siegel, New York.—p. 503.
Miscellaneous Notes on Rat-Bite Fever: Report of Two Cases, Review of American Reports from 1931 to 1940, Observations on Isolation of Spirillum Minus and Results of Standard Serologic Tests During Rat-Bite Fever. P. V. Walley Jr., Portland, Ore.—p. 513.
Beriberi in Male Child Two and One-Half Years of Age. C. G. Kerley and E. J. Lorenze, New York.—p. 526.
Nonhemolytic Streptococcus Omphalitis, Parotitis and Meningitis in Premature Infant: Report of Case. M. J. Hurst and P. S. Astrowe, Kansas City, Mo.—p. 529.
Recovery from Two Attacks of Meningitis Caused by Different Organisms. H. G. Morton and R. W. Roberts, Durham, N. C.—p. 534.
Parental Education in Mental Hygiene. W. H. Missildine, Eagle Grove, Iowa.—p. 536.
Tuberculin Patch Test (Vollmer) on BCG Vaccinated and Control Children. I. S. Neiman, S. R. Rosenthal and W. G. Motel, Chicago.—p. 540.

Cutaneous Reactions to Specific Soluble Substance.—Meyer and Carey used injections of pneumococcus capsular carbohydrate, that is the Francis test, for determining the need of serum therapy in 42 infants and children with typed pneumococcus pneumonia. The results of the test, they say, confirm

its value for controlling the dose of serum in infants and children. However, in their experience the results of the test after chemotherapy with sulfapyridine or sulfathiazole have been irregular and unpredictable. When patients were first treated with serum it was observed that when the result of the Francis test became positive the areas that had been previously injected with specific soluble substance and showed negative results showed typical erythema and wheal reactions after the last dose of serum. Thereafter only one cutaneous test was administered before any serum was given, and the area was observed after each dose of serum. Typical reactions, when they occurred, were usually seen five to fifteen minutes after serum had been administered. It was observed that if single doses of serum were limited to 5 cc. fewer reactions (fever and chills) occurred. In the later cases when the result of the Francis test became positive administration of serum was discontinued. Much smaller amounts of serum were therefore used, with apparently comparable therapeutic effects. The positive reaction is probably due to a local union of antigen and antibody. The time and the intensity of cutaneous reactions varied greatly in the patients given only chemotherapy, but in the patients given serum strong reactions developed and persisted throughout their stay in the hospital.

Effect of Honey on Calcium Retention.—Knott and his associates determined the effect that feeding corn syrup and honey in milk formulas had on the retention of calcium in infants. They found that the average retention of calcium was always higher if honey was included in the formula. The increased retention occurred with both low and high intakes of vitamin D and regardless of the type of milk fed or whether or not lactic acid was used. Further analysis shows that while the favorable influence of honey occurred regardless of other operative factors its magnitude tended to decrease as other beneficial factors were employed. This is to be expected, as each infant has an upper level of optimal response. But despite these factors the use of honey could still improve calcium retention. In view of the results the authors conclude that honey is a carbohydrate which is well suited to the needs of the infant and therefore probably deserves wider use in infant dietaries.

Journal of Pharmacology & Exper. Therap., Baltimore

72:311-426 (Aug.) 1941. Partial Index

Response of Sulfapyridine-Resistant Pneumococci to Hydroxyethylapocupreine. L. H. Schmidt and Clara L. Sesler, Cincinnati.—p. 311.
Studies on Shock Induced by Hemorrhage: I. Effect of Thiamine on Survival Time. W. M. Govier and C. M. Greer, Nashville, Tenn.—p. 317.
Id.: II. Effect of Thiamine on Disturbances of Carbohydrate Metabolism. W. M. Govier and C. M. Greer, Nashville, Tenn.—p. 321.
Histone Zinc Insulin—Its Pharmacologic Characteristics and Its Application in Treatment of Diabetes Mellitus. C. A. Barnes, T. C. Cuttle and G. G. Duncan, Philadelphia.—p. 331.
Quantitative Gastrointestinal Absorption and Renal Excretion of Propylene Glycol. W. Van Winkle Jr., San Francisco.—p. 344.
Production in Cultures Containing Sodium Paranitrobenzoate of a Substance That Affects Action of Sulfathiazole. J. K. Miller, Albany, N. Y.—p. 354.
Effect of Posterior Pituitary Preparations on Large Intestine of Unanesthetized Dog. E. Larson, Philadelphia.—p. 363.
Antagonistic Effect of Asphyxia to Curare Paralysis of Vagus Nerve. H. Mautner and A. Luisado, Waltham, Mass.—p. 386.

72:1-118 (Sept.) 1941. Partial Index

Sweat Response to Drugs with Nicotine-like Action. J. M. Coon and S. Rothman, Chicago.—p. 1.
Physicochemical Properties of Arsphenamines in Relation to Toxicity and Therapeutic Efficiency. H. N. Wright, A. Biedermann, E. Hanssen and C. I. Cooper, Minneapolis.—p. 12.
Local Anesthetic Activity of Certain Derivatives of A and B Naphthol. E. J. Fellows, R. W. Cunningham and A. E. Livingston, Philadelphia.—p. 27.
Differential Excretion of Bromide and Chloride Ions and Its Role in Bromide Retention. O. Bodansky and W. Modell, New York.—p. 51.
Studies of Toxicity and Pharmacology of Pantothenic Acid. K. Unna and J. G. Greslin, Rahway, N. J.—p. 85.
Studies on Relation of Drug Addiction to Autonomic Nervous System: Results of Cold Pressor Tests. C. K. Himmelsbach, Lexington, Ky.—p. 91.
Pain Threshold Measurements in Dog. H. L. Andrews and W. Workman, Lexington, Ky.—p. 99.
Quantitative Comparisons of Activity of Sulfanilamide, Sulfapyridine, Sulfathiazole and Sulfadiazine Against Escherichia Coli in Vivo and in Vitro. H. J. White, J. T. Litchfield Jr. and E. K. Marshall Jr., Baltimore.—p. 104.

Journal of Thoracic Surgery, St. Louis

11:1-130 (Oct.) 1941

Pulmonary Abscess: Specific Method of Treatment Applicable to Both Acute and Chronic Lesions. F. B. Gurd, Montreal, Canada.—p. 1.
Anatomic Changes in Lungs Following Thoracoplasty: Study of 134 Autopsy Cases. O. Auerbach, Staten Island, N. Y.—p. 21.
*Surgery in Pulmonary Tuberculosis: Study of Collapse Therapy, with Special Reference to Thoracoplasty, Based on Results of Thirteen Years. R. Adams, Boston, and P. Dufault, Rutland, Mass.—p. 43.
*Thoracoplasty for Tuberculosis: Late Results. G. F. Skinner, L. Macpherson and Irene Allen, St. John, N. B., Canada.—p. 54.
Analysis of 100 Consecutive Cases of Thoracoplasty with No Operative Mortality. R. H. Dieffenbach, Newark, N. J., and A. D. Creeca, Verona, N. J.—p. 65.
Analysis of 104 Cases of Thoracoplasty for Pulmonary Tuberculosis. G. G. Finney, Baltimore.—p. 76.
Results of Thoracoplasty. H. Meltzer, Ninette, Man., Canada.—p. 84.
Thoracoplasty in Bilateral Pulmonary Tuberculosis. A. M. Vineberg, D. Ackman and M. Aronovitch, Montreal, Canada.—p. 95.
Results in Ninety Consecutive Thoracoplasties for Pulmonary Tuberculosis. A. H. Aufses, New York.—p. 98.
Rest and Collapse in Pulmonary Tuberculosis. D. Salkin and A. V. Cadden, Hopemont, W. Va.—p. 109.
New Classification of Pulmonary Tuberculosis. D. Salkin and A. V. Cadden, Hopemont, W. Va.—p. 126.

Surgery in Pulmonary Tuberculosis.—Adams and Dufault review the results following thoracoplasty on 241 consecutive patients submitted to operation during thirteen years. The last patient was operated on at least two years ago. Among the group considered there were 22 with tuberculous and mixed empyema. Of the entire group 144 were apparently cured. The tuberculosis of 11 was arrested, of 2 was apparently arrested, of 27 was quiescent, of 10 was unimproved and of 1 it was worse. The status of 8 is unknown, and of the 33 patients who died 28 did so from tuberculosis, 7 from empyema and 3 from a nontuberculous disease. Twelve with empyema were cured, 1 improved, 2 were worse or unimproved and 7 died.

Thoracoplasty for Tuberculosis.—According to Skinner and his associates, 226 patients had thoracoplasty between 1924 and 1941; 100 had it by the end of 1935. Of 50 operated on before 1933 24 are apparently still cured in 1941, 5 are improved, 2 are unimproved and 19 are dead. Of the 50 operated on during 1933, 1934 and 1935 30 are apparently cured in 1941, 2 are improved, 5 are unimproved and 13 are dead. Only a preliminary report is possible for the 126 patients operated on since 1935, but up to date 58 are apparently cured, 43 are improved, 17 have unsatisfactory results and 8 are dead.

New York State Journal of Medicine, New York

41:1891-1986 (Oct. 1) 1941

Soldier's Heart. L. F. Bishop Jr., New York.—p. 1915.
*Evaluation of Surgical Treatment of Hypertension. G. J. Heuer and F. Glenn, New York.—p. 1922.
Control of Pain and Discomfort by Subcutaneous Injection of Oxygen. J. H. Evans, Buffalo.—p. 1927.
Differential Diagnosis of Conditions in Upper Part of Abdomen. M. A. Ramirez, New York.—p. 1934.
Cryptitis—Perianal and Perirectal Infections. F. L. Sullivan, Scotia.—p. 1940.
Cigaret Smoking in Pregnancy. F. J. Schorneck, Syracuse.—p. 1945.
The Part of the Private Pediatrician in the School Health Program. W. E. Ayling, Syracuse.—p. 1949.

41:1987-2082 (Oct. 15) 1941

New Medical Attack on So-Called "Mental" Disease. F. Kennedy, New York.—p. 2013.
Industrial Health: Periodic Examination. M. Woody, New York.—p. 2018.
Id.: New Medical Opportunities in National Defense Industries. C. D. Selby, Detroit.—p. 2023.
Industrial Health and the General Practitioner. L. D. Bristol, New York.—p. 2026.
Clinical Studies in Electrocardiography: IV. Value of Electrocardiogram in Coronary Thrombosis, with Special Reference to Localization of Infarct. A. Lieberson, J. Chasnoff and A. A. Goldbloom, New York.—p. 2032.
Incidence of Heart Disease in University Student Age Group. Muriel Cuykendall, Ithaca.—p. 2037.
Medical Diagnosis of Mental Defect. E. J. Humphreys, Thiells.—p. 2041.

Surgical Treatment of Hypertension.—Heuer and Glenn studied the results of operative procedures (19 anterior root sections, 12 supradiaphragmatic splanchnicotomies and 18 splanchnic resections with interruption of the first and second

lumbar ganglions) on 49 hypertensive patients. Such therapy is indicated because (1) a lowering of blood pressure has followed the operative procedures, (2) changes in the fundi have occurred and in many cases persisted, (3) headaches have been relieved for variable periods, (4) nervousness has been diminished and (5) early fatigue has been lessened. Twenty-six of the patients are alive from two months to six years and nine months after operation.

North Carolina Medical Journal, Winston-Salem

2:527-578 (Oct.) 1941

First Ten Years of Duke University School of Medicine and Duke Hospital. W. C. Davison, Durham.—p. 527.
The Medical Profession and the Problem of Mental Disorder. J. Watson, Raleigh.—p. 532.
Public Health and National Defense. M. J. Rosenau, Chapel Hill.—p. 537.
Tumors of Larynx and Hypopharynx. L. H. Clerf, Philadelphia.—p. 539.
Appendicitis in Children. T. M. Watson, Greenville.—p. 541.
Practical Plan for Use of Vitamin K in Prevention of Hemorrhage in Newly Born Infant. R. B. Lawson, Chapel Hill.—p. 544.
Delayed Relaxation of Tendon Reflexes as Aid in Diagnosis of Myxedema. G. T. Harrell, Winston-Salem, and D. Daniel, Durham.—p. 549.
Health Problems in Area Surrounding Large Military Establishment. M. T. Foster, Fayetteville.—p. 552.
Cardiac Decompensation in Congenital Heart Disease: Report of Case. Dorothy B. Wyvell, Durham.—p. 557.

Northwest Medicine, Seattle

40:351-396 (Oct.) 1941

Diabetes: Disturbance in Endocrine Regulation of Blood Sugar; Fundamentals of Physiology of Diabetes, Carbohydrate Utilization and Carbohydrate Tolerance; Endocrine Balance in Carbohydrate Metabolism. S. Soskin, Chicago.—p. 356.
Effect of Anterior Pituitary-like Substance on Carbohydrate Metabolism. B. Vidgoff and Rosa Kubin, Portland, Ore.—p. 361.
Indications for Gastroscopy. P. B. Nutter, Spokane, Wash.—p. 363.
Symptomless Period of Bronchial Foreign Bodies. P. Bailey, Portland, Ore.—p. 365.
Influence of Suggestion on Size of Bronchial Lumen: Bronchoscopic Study and Report of One Case. W. B. Faulkner Jr., San Francisco.—p. 367.
Cancer from Pathologist's Angle. W. Dock, New York.—p. 369.
Cosmetic and Functional Aspects of Bone Grafting in Mandibular Fractures. C. M. MacKenzie, Spokane, Wash., and D. H. Sharpless, Everett, Wash.—p. 372.

Oklahoma State Medical Assn. Jour., Oklahoma City

34:419-464 (Oct.) 1941

Foreign Bodies of Air and Food Passages: Some Observations on Series of 220 Cases. L. C. McHenry, Oklahoma City.—p. 419.
Value of Frei Antigen of Chick Embryo Origin in Diagnosis of Lymphogranuloma Venereum. R. B. Greenblatt, Augusta, Ga.—p. 423.
The Practicing Physician and the Public Health Department. J. F. Hackler, Oklahoma City.—p. 425.
Hoarseness. G. L. Tracewell, Okmulgee.—p. 427.
Corneal Injuries and Complications. W. W. Mall, Ponca City.—p. 429.
Psychosomatic Medicine. H. M. Galbraith, Oklahoma City.—p. 431.

Philippine Medical Association Journal, Manila

21:423-480 (Sept.) 1941

Tañgan-Tañgan Oil as Purgative for Sick Infants and Children. R. Guevara and A. P. Jongco, Manila.—p. 423.
Observations on Use of Sulfapyridine in Treatment of Pneumococcic Pneumonia Among Philippine Army Personnel. V. Luna, A. T. Cruz and C. B. Icasiano, Quezon, Tayabas.—p. 435.
Concept of Focal Infection in Ophthalmology. A. R. Ubaldo and G. de Ocampo, Manila.—p. 443.
*Encephalopathia Saturnina: Report of Case. J. Santillan, F. S. Soriano, Manila, and J. S. Ongjoco, Pasay.—p. 455.

Encephalopathia Saturnina.—Santillan and his associates believe that they are apparently reporting the first case of encephalopathia saturnina to occur in the Philippines. The patient started working in a large printing establishment in 1909. During the time of his connection with the firm he was always in contact with lead. He remembers that about two years after he was employed he began to have periodic attacks of dizziness and headache. He recalls two attacks of abdominal colic. After nineteen years of continuous work he had a sudden and severe convulsive attack. This attack was followed by loss of consciousness, and immediately after it he was unable to talk or walk. He was obliged to stop working. Gradually, while at home, he recovered his speech, but even after twelve years without contact with lead he has never regained the full use of his right upper and lower extremities. The symptoms coincide with those described by Tanquerel des Planches and Westphal in lead encephalopathy of the apoplectic form. Lead poisoning, like all other occupational diseases, is compensable. The patient was given compensation for total and permanent disability.

Physiological Reviews, Baltimore

21:529-632 (Oct.) 1941

Factors Affecting the Tests of Kidney Function. R. C. Herrin, Madison, Wis.—p. 529.
Anaphylaxis. C. A. Dragstedt, Chicago.—p. 563.
Antihormones. K. W. Thompson, New Haven, Conn.—p. 588.

Psychiatric Quarterly, Utica, N. Y.

15:621-874 (Oct.) 1941. Partial Index

Critical Analysis of Insulin Therapy at Rochester State Hospital. W. Libertson, Rochester, N. Y.—p. 635.
The Retina and Ocular Tension During Prolonged Insulin Coma, with Autopsy Eye Findings. A. Gralnick, Central Islip, N. Y.—p. 648.
Some Observations on Cardiovascular Changes in Shock Therapy. H. Cleckley and D. B. Eggleston Jr., Augusta, Ga.—p. 662.
Electrocardiogram in Metrazol Therapy. L. Wender and A. Jezer, Hastings-on-Hudson, N. Y.—p. 680.
*Report on Subconvulsive Reaction to Electric Shock and Its Sequelae in Normal Subject. C. Watkins, E. J. Stainbrook and H. Löwenbach, Raleigh, N. C.—p. 724.
Electric Shock Therapy in Psychoses: Convulsive and Subconvulsive Methods. S. Androp, Catonsville, Md.—p. 730.
Artificial Fever Therapy in General Paresis with Electroencephalographic Studies. A. E. Bennett, P. T. Cash and C. S. Hoekstra, Omaha.—p. 750.
Results Obtained from Administration of 12,000 Doses of Metrazol to Mental Patients: Preliminary Report. M. Zeifert, Brooklyn.—p. 772.
Relation of Group of Highly Improved Schizophrenic Patients to One Group of Completely Recovered and Another Group of Deteriorated Patients. O. Kant, Worcester, Mass.—p. 779.
Mapharsen in Treatment of Therapeutic Benign Tertian Malaria. D. Whitehead and J. J. Dorey, Utica, N. Y.—p. 790.
Rorschach Method as Prognostic Aid in Insulin Shock Treatment of Schizophrenics. Z. A. Piotrowski, New York.—p. 807.

Subconvulsive Reaction to Electric Shock.—Watkins and his associates point out that a volunteer, a physician of 25, was submitted to an electric subconvulsive shock. His behavior and experience during recovery were similar to those observed in patients with mental disease after convulsive shocks. The principal features of the volunteer's behavior were absence of somatic and mental complaints, amnesia and disorientation.

Public Health Reports, Washington, D. C.

56:1941-1980 (Oct. 3) 1941

Our Inadequate Treatment of the Mentally Ill as Compared with Treatment of Other Sick People. V. H. Vogel.—p. 1941.
*Dermatitis from Cutting Oils. L. Schwartz.—p. 1947.
*Lead and Arsenic Content of Urines from Forty-Six Persons with No Known Exposure to Lead or Arsenic. S. H. Webster.—p. 1953.
*Rat-Bite Fever in Washington, D. C., Due to Spirillum Minus and Streptobacillus Moniliformis. C. L. Larson.—p. 1961.

Dermatitis from Cutting Oils.—Schwartz points out that soluble and insoluble cutting oils and greases used for lubricating and preserving the temper of cutting tools are the most frequent causes of dermatitis among machinists and metal workers. All petrolatum oils have a defatting action on the skin. Consequently a greasy skin with active sebaceous glands is less apt to be defatted than a dry skin. The comedos, folliculitis and boils caused by cutting oils and greases can be successfully treated and prevented by a daily change of work clothes and frequent washing of the affected parts. Pus should be evacuated surgically, and the judicious use of compresses of solutions of boric acid, mercury bichloride 1:1,000 or potassium permanganate 1:2,000 will usually clear up the condition. The allergic types of cutting oil dermatitis are best treated by removing the affected workers from contact with the offending agents. Antiseptic lotions should be used for the moist types; ointments for the dry chronic types; dressings of boric acid, Burow's solution or mercury bichloride 1:1,000 for the acute moist types, and boric acid ointment, zinc ointment, Lassar's paste and coal tar preparations for the dry or dry eczematoid types of dermatitis. Workers with dry, defatted hands should apply a vegetable or animal fat ointment before and after work.

Urinary Content of Lead and Arsenic of Unexposed Persons.—Webster determined the urinary lead and arsenic concentration of the morning specimens of 46 persons (28 adults and 18 children) with no known exposure to lead arsenate. A wide concentration range was found. However, there was no uniformity among the different members of a given family. There was a close correspondence between specific gravity and phosphate concentration. Comparison of these lead and arsenic values with those of men and women living in an apple-growing locality with potential exposure to lead arsenate from the ingestion of apples having the residue from lead arsenate spray on their surfaces and/or from inhalation of lead arsenate dust, spray or particulate matter revealed about the same average values, but the averages were significantly lower for the groups not residing in the apple-growing district. No evidence was discovered to indicate that the urinary lead and arsenic content of children differs from that of adults.

Rat-Bite Fever in Washington, D. C.—Larson reports 3 cases of rat-bite fever observed in Washington, D. C.; from 1 patient Leptospira morsus muris and from 2 Streptobacillus moniliformis were isolated. The 3 cases furnish the first record of isolation of L. morsus muris from a patient in Washington and also the only instances of infection with Streptobacillus in the locality. From these cases the author finds that the clinical diagnosis of rat-bite fever may be difficult when certain cardinal features are absent, but this can be offset by employing proper laboratory methods for confirmation.

86:1981-2032 (Oct. 10) 1941

Doctors' Calls in Connection with Illness from Specific Diseases Among 9,000 Families, Based on Nationwide Periodic Canvasses, 1928-1931. Selwyn D. Collins.—p. 1981.
*Ornithodorus Hermsi and Relapsing Fever in Oregon. G. E. Davis. —p. 2010.

Ornithodorus Hermsi and Relapsing Fever in Oregon.—Davis points out that 2 cases of proved and 3 of suspected relapsing fever have been encountered in central Oregon. He collected 52 specimens of Ornithodorus hermsi from a hollow pine log in the immediate area in which the infection occurred. The ticks were tested in seven groups of 5 each and in three groups of 4, 6 and 7 each by allowing them to feed on white mice. Spirochetes were recovered from 6 of 10 test mice. The presence of relatively large numbers of O. hermsi in Douglas firs in Colorado and in the pine log in Oregon suggests that dwellings could be infested and that contact with decaying wood used as fuel or for lumber may be a source of infection.

Radiology, Syracuse, N. Y.

37:391-520 (Oct.) 1941

Intrathoracic Neurogenic Tumors. K. Kornblum and H. H. Bradshaw, Philadelphia.—p. 391.
Silicosis. G. Clement, Duluth, Minn.—p. 407.
Factors Involved in Production and Development of Silicotic Lesions. W. S. Lemon, Rochester, Minn.—p. 413.
*Twelve Year Review of X-Ray Therapy of Gas Gangrene. J. F. Kelly and D. A. Dowell, Omaha.—p. 421.
*Roentgen Radiation in Experimental Clostridium Welchi Infection (Gas Gangrene) in Dogs: Preliminary Report. A. H. Dowdy and L. L. Sewell, Rochester, N. Y.—p. 440.
Influence of Emotions on Esophageal Function: Comparison of Esophagoscopic and Roentgenologic Findings. W. B. Faulkner Jr., F. H. Rodenbaugh and J. R. O'Neill, San Francisco.—p. 443.
Acute (Primary) Coccidioidomycosis: Roentgen Findings in Group "Epidemic." R. A. Powers and Dorothy J. Starks, Palo Alto, Calif. —p. 448.
Roentgen Diagnosis of Neurinoma of Thorax. I. N. Odessky, Moscow, Soviet Union.—p. 454.
Carcinoma of Male Breast. M. D. Sachs, Portland, Ore.—p. 458.
Oral Carcinoma. W. E. Howes and L. Bernstein, Brooklyn.—p. 468.
Depth Dose Calculation Wherein Absorption in Unit Volume in Depth, i. e., Volume Depth Dose, Is Index of Effective Radiation. W. H. Meyer, New York.—p. 476.
Cerebral Arteriography with Diodrast, Fifty per Cent. S. W. Gross, New York.—p. 487.
*Rhomboid Depression of Clavicle. S. Shulman, Far Rockaway, N. Y. —p. 489.

Roentgen Therapy for Gas Gangrene.—Kelly and Dowell assemble the data on twelve years of the roentgen treatment of gas gangrene. The decrease in mortality, they say, indicates that with irradiation the disease need no longer be considered serious. Two of their first 8 patients so treated died, but of 364 patients from all sources who were treated during the twelve years (ending with May 1940) with one or more roentgen treatments, 42 died, a mortality of 11.5 per cent. The mortality for 288 of the 364 patients given three or more treatments was 5.9 per cent. The diagnosis of the disease in 93 was based on clinical grounds, in 41 on clinical grounds and roentgen evidence of gas in the tissues and in 207 on laboratory evidence of Clostridium welchi, and the method of diagnosis for the remaining 23 was not stated. The number of deaths in these four groups was, respectively, 16, 7, 18 and 1. From an analysis of the data the authors conclude that the roentgen treatment of gas gangrene has a direct effect in preventing and curing the infection, and that (1) the administration of serum appears unnecessary, (2) irradiation is effective after serum therapy fails, (3) giving serum may even be harmful to the diabetic patient in whom gas gangrene develops, (4) giving serum may be harmful to aged patients and (5) roentgen therapy of gas gangrene has completely eliminated the necessity of extensive surgical intervention. Amputation and extensive débridement are seldom necessary. They should never be necessary if proper treatment is started early. The study stresses the need for the general use of roentgen therapy for inflammatory disease at the bedside, with an apparatus of adequate kilovoltage. Acute spreading peritonitis has been successfully treated by irradiation. Sulfanilamide should not be used during the roentgen treatment of acute inflammatory processes. The curative action of the roentgen rays is apparently due to the rays' nonspecific antitoxic effect. This antitoxic effect, resembling the nonspecific effect in preventing peritonitis after colonic and rectal operations, was first reported by the Ford Hospital group.

Gas Gangrene in Dogs.—Dowdy and Sewell produced a syndrome comparable to that of Clostridium welchi disease in man in 66 dogs. Irradiation had some therapeutic effect in combating the infection in 25 dogs as compared with 25 controls. The survival rates were 36 and 20 per cent, respectively. Preliminary studies on the effect of the time of treatment, the amount of treatment, the size of the dose and the kilovoltage show that they all have a bearing on the survival rate.

Rhomboid Depression of Clavicle.—Shulman calls attention to a normal anatomic landmark of the clavicle which ordinarily is not visible on the roentgenogram but which occasionally may be so well developed as to simulate destructive lesions of bone. It is important to differentiate such an anomaly from the various pathologic conditions (osteomyelitis, tuberculosis, syphilis and malignant neoplasms) known to involve the region of the clavicle. A comprehensive survey of the American literature disclosed only a single reference to the possible occurrence of this anomaly. Occasionally the rhomboid depression may be extremely deep and penetrate about halfway through the thickness of the clavicle. The outline of the upper margin of the defect is usually irregular, and its density is slightly increased. Under such conditions, the anomalous development of the depression appears as a large osseous defect and may easily be mistaken for a destructive process.

Review of Gastroenterology, New York

8:343-420 (Sept.-Oct.) 1941

Electrocardiographic Changes Following Hematemesis in Peptic Ulcer. D. Scherf, H. Reinstein and S. D. Klotz, New York.—p. 343.
Pathogenesis of Peptic Ulcer. I. W. Held, New York.—p. 350.
Ulcerative Colitis and Its Management. M. Kraemer, Newark, N. J. —p. 357.
Simulation of Bleeding Penetrating Duodenal Ulcer. M. Weingarten, New York.—p. 367.
Underweight: Problem in Treatment. H. A. Monat, Washington, D. C. —p. 371.
Management and Treatment of Acute Food Poisoning. T. G. Simonton, Pittsburgh.—p. 376.
Gastric Mucin Silver Iodide in Rectal and Colonic Therapy. A. G. Dujat, New York.—p. 381.
Remarks on "Material" and "Formal" Causes of Cancer. S. P. Reimann, Philadelphia.—p. 385.
X-Ray Diagnosis of Gastrointestinal Malignancy. J. S. Lehman, Philadelphia.—p. 386.
Surgery in Gastrointestinal Cancer. W. L. Martin, Philadelphia.—p. 387.
Cancer of Colon. N. V. Ludwick, Philadelphia.—p. 388.
Melanosis Coli. L. H. Block, Chicago, and B. L. Greene, Elgin, Ill.—p. 393.
Primary Carcinoma of Head of Pancreas: Clinical Observations. J. Friedenwald and T. H. Morrison, Baltimore.—p. 400.
Ruptured Peptic Ulcer Producing Jaundice by Pressure on Common Bile Duct. L. C. Kelly, New York.—p. 408.

Texas State Journal of Medicine, Fort Worth

37:331-386 (Sept.) 1941

Present Status of Surgical Procedures on Biliary Tract. R. R. Graham, Toronto, Canada.—p. 335.
Surgical Treatment of Ureteral Stones. H. M. Spence, Dallas.—p. 340.
Plastic Construction of Artificial Vagina. T. G. Blocker Jr., Galveston.—p. 345.
Differential Diagnosis of Internal Derangement of Knee. W. A. Bishop Jr., Wichita Falls.—p. 348.
Etiology and Symptomatology of Brucellosis. D. Neighbors, Fort Worth.—p. 353.
Diagnosis of Brucellosis. F. T. McIntire, San Angelo.—p. 355.
Brucellosis, a Public Health Problem. L. L. Terry, Galveston.—p. 359.
Contracted Pelvis and Delayed Labor. E. D. Plass, Iowa City.—p. 363.
Hypertrophic Pyloric Stenosis in Infants: Result of Birth Injury. J. G. Flynn, Houston.—p. 367.
Ephedrine Sulfate Intranasally as Cause of Uterine Bleeding. Evelyn Gass Powers, Amarillo.—p. 369.
Roentgen Examination as Diagnostic Procedure in Hemorrhage from Gastrointestinal Tract. G. W. Holmes, Boston.—p. 372.

37:387-448 (Oct.) 1941

Aims and Methods of Texas Pneumonia Control Program. J. Kopecky, San Antonio.—p. 391.
Modern Study of Pneumonia Patient. W. G. Maddox, Dallas.—p. 393.
Recent Advances in Treatment of Pneumonia. C. T. Stone, Galveston.—p. 396.
Some Economy and Efficiency Devices in Tuberculosis Control Work. J. E. Johnson, Mineral Wells.—p. 400.
Common Neurosurgical Operations for Relief of Pain. R. C. L. Robertson, Houston.—p. 404.
Diabetes and Pregnancy. J. A. Clapp Jr., Houston.—p. 408.
Management of Sterility in Female. R. E. Lee, Dallas.—p. 414.
X-Ray Therapy in Asthma and Hay Fever. C. F. Crain, Corpus Christi.—p. 419.
Intraoral Radiation Therapy. P. E. Wigby, Houston.—p. 422.
Reading Problems, Their Causes and Effects. L. H. Quinn, Dallas.—p. 428.
Relation Between the Radiologist and the Referring Physician. R. T. Wilson, Austin.—p. 433.

Western J. Surg., Obst. & Gynecology, Portland, Ore.

49:527-598 (Oct.) 1941

*Uterine Fibroids with Pregnancy. G. G. Thompson, Seattle.—p. 527.
Bronchoscopy as Diagnostic Aid in Nontuberculous Pulmonary Disease. L. H. Clerf, Philadelphia.—p. 557.
Ectopic Pregnancy in Hawaii. O. L. Schattenburg, Honolulu, Hawaii.—p. 562.
Pregnancy Tests of Past and Present. E. Henriksen, Los Angeles.—p. 567.
Compression Fractures of Dorsolumbar Vertebrae. J. W. Gullikson and E. R. Anderson, Tacoma, Wash.—p. 576.
Spontaneous Amputation of Cervix During Labor. R. D. Reekie, Spokane, Wash.—p. 581.
Action of Iodine on Goiter and Action of Thyroactivator in Pathogenesis of Primary Goiter: Clinical, Laboratory and Experimental Observations. S. Brock, Chicago.—p. 585.

Uterine Fibroids with Pregnancy.—From a study of one hundred and forty-four labors among 118 women with uterine fibromyoma whom Thompson encountered among two thousand consecutive private deliveries he concludes that fibroids do not negate pregnancy or nullify the chance of viable infants. The history (previous pregnancies, abortions, labors, menstruation and symptoms) seems the most important indicator, with the size of the fibroids a close second: The better the history the better the course, and the larger the fibroid the poorer the course. There was an increased incidence (10.8 per cent) of premature rupture of the membranes, premature labor, placenta previa, breech presentation and hemorrhage during pregnancy and labor and immediately post partum among the 118 women. Pain and tenderness during pregnancy indicated trouble. However, the trouble was grave in only a few women, and it was usually heralded and never calamitous or catastrophic for the mother. Therefore it seems that without the most serious deliberation a woman should never be informed that pregnancy is impossible because of fibroids and that a miscarriage (which occurred in only 6 among the group) will occur or that much trouble awaits her if she becomes pregnant. Likewise the removal of the uterus with the fibroids should never be urged or suggested—especially in a woman who desires children. The woman should be encouraged to keep her troublesome organ so that it may fulfil its physiologic function.

FOREIGN

An asterisk (*) before a title indicates that the article is abstracted below. Single case reports and trials of new drugs are usually omitted.

British Journal of Radiology, London

14:307-342 (Oct.) 1941

Value of Cooperation Between the Radiologist and the Gastroscopist. S. W. Davidson and J. D. Rose.—p. 307.
Changes in Bones, Joints and Soft Tissues Associated with Disease or Injury of Central Nervous System. J. F. Brailsford.—p. 320.
Mucous and Salivary Gland Tumors: Report on Series of Fifty-Seven Cases, with Special Reference to Radium Treatment. M. Lederman.—p. 329.
Construction of Universal Dosemeter with Notes on Its Use for Measurement of High Resistances. F. Happey.—p. 336.

British Journal of Surgery, Bristol

29:1-164 (July) 1941

Postoperative Complications of Pulmonary Lobectomy: Clinical and Experimental Study. F. J. S. Gowar.—p. 3.
Osteitis of Metatarsal Sesamoid Including Report of Case of Acute Pyogenic Osteomyelitis. R. Smith.—p. 19.
Coxa Plana, with Special Reference to Its Pathology and Kinship. H. J. Burrows.—p. 23.
Exomphalos: Radical Cure in Infant, Twenty Minutes Old. L. Rogers.—p. 37.
Thoracicoabdominal Gastrectomy for Carcinoma of Thoracic Stomach. R. W. Raven.—p. 39.
Earliest English Example of Bilateral Cervical Rib. A. J. E. Cave.—p. 47.
Congenital Atresia of Bile Ducts. J. T. Chesterman.—p. 52.
Surgical Treatment of Syringomyelia. C. Worster-Drought, C. P. G. Wakeley and J. Shafar.—p. 56.
Leontiasis Ossea. G. K. Kirkland.—p. 74.
One Stage Operation for Hypospadias. G. Humby, foreword by T. T. Higgins.—p. 84.
*Ischiofemoral Arthrodesis. H. A. Brittain.—p. 93.
Collateral Venous Circulation in Case of Thrombosis of Inferior Vena Cava and Its Embryologic Interpretation. J. A. Keen.—p. 103.
Intracapsular Fractures of Neck of Femur: Final Results of Seventy-Five Consecutive Cases Treated by Closed Method of Pinning. A. L. Eyre-Brook and K. H. Pridie.—p. 115.
Tumors of Small Intestine. J. E. Morison.—p. 139.

Ischiofemoral Arthrodesis.—Brittain used ischiofemoral arthrodesis for 35 patients with tuberculosis, osteoarthritis or infective arthritis of the hip. Ischiofemoral arthrodesis afforded a high percentage of fusions in his patients. The operation can be offered any patient less than 65, provided that in tuberculosis the disease does not extend down into the ischium. Ischiofemoral arthrodesis is fusion between the ischium and the shaft of the femur in the region of the small trochanter. The operation consists in performing a subtrochanteric osteotomy at a previously selected site, passing the osteotome into the ischium in the same line for 1 inch (2.5 cm.) and introducing a massive flat tibial bone graft, that is the entire medial surface of the tibia. One end of the graft is well implanted in the ischium and the other between the two fragments of the osteotomy. The limb is then abducted so that the lower fragment comes into contact with the ischium, and thus the graft is supported throughout its entire length. The one possible disadvantage to ischiofemoral arthrodesis, as to the iliofemoral operation, is that the disease may involve the graft. Bony fusion followed the first operation in 80 per cent of the author's patients. For 3 patients the operation had to be repeated; for 1 of these it was performed three times. The results with these 3 patients bring the percentage of fusions to 88. If fusion should not occur because the graft was passed too high into the diseased area, the operation can be repeated.

British Medical Journal, London

2:465-496 (Oct. 4) 1941

Head Injuries in Motor Cyclists: Importance of Crash Helmet. H. Cairns.—p. 465.
Congenital Bilateral Renal Hypoplasia, with Short Review of Literature: Case. J. E. Murray and R. A. Sandison.—p. 471.
*Review of Dyspepsia in Army. C. A. Hinds-Howell.—p. 473.
Crush Injury with Renal Failure and Recovery. G. Blackburn and W. W. Kay.—p. 475.
*Bacteriology of Air Raid Wounds Examined Within Forty-Eight Hours of Infliction. E. T. C. Spooner.—p. 47.
War Conditions and the Mental Health of the Child. F. Bodman.—p. 486.

Dyspepsia in the Army.—Hinds-Howell cared for 804 patients with dyspepsia from October 1939 to December 1940. The symptoms of 80, or only 10 per cent, originated during service in the army; in 13 of the 80 the character of the symp-

toms was neurotic. The symptoms of 47 of the 91 Canadian patients developed after their arrival in England. Six patients were New Zealand soldiers. Of the 707 British patients 391 (55 per cent) presented definite evidence of peptic ulceration. The highest incidence of symptoms of duodenal ulcer was among soldiers between 20 and 30 years of age; the same was true of gastric ulcer, although the curve was flatter. Thirty-five had had an acute hemorrhage, 34 a perforated peptic ulcer and 29 an appendectomy, but the duodenal symptoms were relieved only for about two years. The onset of pain after meals was unreliable as a diagnostic factor. Vomiting, more common in soldiers than in civilians, was almost universal, and usually it was followed by partial or complete relief of symptoms. Men with proved ulcers are unfit for military service, but officers and occasionally noncommissioned officers or other useful men should be retained for home duty if arrangement for a suitable diet can be made.

Bacteriology of Recent Air Raid Wounds.—To determine whether pathogenic bacteria are present in wounds immediately after their infliction Spooner examined material (pieces of external wounds, wound tracks and foreign bodies from thirty-one early air raid wounds) at the first surgical cleansing. Pyogenic cocci were less frequent and coagulase-negative staphylococci, aerobic spore-bearing bacilli and Clostridium welchi were more common in recent than in older wounds. No bacteria were found in six wounds. Other bacteria found were Escherichia coli in six, diphtheroid bacilli in three, nonhemolytic streptococci in three, Streptococcus viridans in two, micrococci in six and other clostridiums in one.

Guy's Hospital Reports, London

90:73-298 (Nos. 2, 3 and 4) 1940-1941. **Partial Index**

Astley Cooper and Arterial Surgery. R. C. Brock.—p. 104.
*Cirsoid Aneurysm. R. Davies-Colley.—p. 134.
*Cirsoid Aneurysm of Chest Wall. N. L. Eckhoff.—p. 141.
Angiomatous Malformations of Brain. D. W. C. Northfield.—p. 149.
*Traction Rupture of Great Vessels of Chest Following Injury to Head. K. Simpson and A. Cooper.—p. 196.
Experiences in Pulmonary Artery Ligation. R. C. Brock.—p. 217.
Popliteal Aneurysm: Report of Bilateral Case Treated by Bilateral Excision. A. G. Hardy and H. E. H. Denham.—p. 244.

Cirsoid Aneurysm.—Davies-Colley reports the 6 instances of cirsoid aneurysm (only 3 were of the head) that he encountered in the last twenty years. In discussing their probable pathogenesis he states that all cirsoid aneurysms are the result of a developmental defect in the formation of the capillary barrier between arteries and veins. Most of them follow an intermediate stage of visible dilatation of the capillaries, the cavernous angioma. Trauma may play the part of an accessory factor in their formation but is never the only cause.

Cirsoid Aneurysm of Chest Wall.—The large cirsoid aneurysm of the wall of the chest that Eckhoff discusses had feeding vessels around the periphery, and one enormous vessel (not discovered until the third operation) entered from behind through an intercostal space. Whereas surgical measures were finally successful in causing the pulsation to cease, a capillary angioma appeared and spread well beyond the original confines of the aneurysm. Irradiation caused the angioma to regress. After this an ulcerated area appeared which was resistant to all ordinary methods of treatment, including cutaneous grafting, for a long time. This patient had a dense mass of scar tissue between the wall of the chest and the surface of the skin. The two treatments with radium added to the scar tissue. Healing followed excision of the whole ulcerated area, the removal of all scar tissue and the covering of the area with a large razor graft.

Journal of Hygiene, London

41:101-224 (Sept.) 1941. **Partial Index**

Intratracheal Inoculation of Anthracite Dust Mixed with Dead Human Tubercle Bacilli into Rabbits. S. L. Cummins.—p. 101.
Bacterium Dysenteriae Sonne: Study of Forty Strains, with Particular Reference to Appearance of Colonies. A. J. Rhodes and Christine Reid.—p. 105.
Effect of Humidity of Air on Disinfection Capacity of Mechanically Atomized and Heat Volatilized Germicidal Aerosols. A. H. Baker and C. C. Twort.—p. 117.
Tests of Significance of Differences in Degree of Pollution by Coliform Bacteria and on Estimation of Such Differences. H. J. Buchanan-Wollaston.—p. 139.
Physical Type in Pneumoconiosis. E. A. Aslett, P. D. Hart, W. J. Martin and W. T. Russell.—p. 169.

Lancet, London

2:387-414 (Oct. 4) 1941

*Influenza in England in 1940-1941. C. H. Andrewes, R. E. Glover, N. P. Hudson, Dora Lush and C. H. Stuart-Harris.—p. 387.
Extraoral Splinting of Edentulous Mandible. L. Pohl.—p. 389.
External Pin Fixation for Fractures of Mandible. R. Mowlem, A. B. MacGregor, J. L. D. Buxton and J. N. Barron.—p. 391.
Blood Changes Following Controlled Hemorrhage in Man. J. Wallace and E. P. Sharpey-Schafer, with technical assistance of A. C. Pincock.—p. 393.
Trichiniasis in South East London. E. Davis and E. N. Allott.—p. 396.
Trichiniasis with Cerebral Symptoms: Report of Two Cases. J. C. Houston and L. Ross.—p. 397.

Influenza in England.—According to Andrewes and his associates, the outbreak of respiratory disease prevalent in southern England in the spring of 1940 was of the long drawn out rather than of the sharply peaking form, "clinically typical" influenza was relatively infrequent and tracheobronchitis with slow recovery was prevalent. Garglings from patients with a diagnosis of clinical influenza were tested by the intranasal inoculation of ferrets, but no evidence was obtained that that virus was prevalent. None of the garglings produced infection in the ferrets. Although there was no general epidemic of influenza in the winter of 1940-1941, clinical influenza occurred sporadically in London and elsewhere in southern England. Eight of 19 garglings or samples of serum received in January and February from as many patients provided evidence that the disease was influenza A. During the same period a detailed clinical study of the acute respiratory infections occurring in a military hospital in southern England was being made, and although no actual epidemic was observed there was a sharply increased incidence of influenza in January and February. The influenza of many of the patients was similar to that seen in 1937 and 1939. Pathologic investigations revealed that 79 per cent of the local material studied was of virus A influenza. Not one of the specimens from 16 patients from various districts examined during March and April 1941 gave evidence that influenza A was concerned. The 1940-1941 studies have shown that testing serums for neutralizing antibodies is a far more delicate index of infection than the inoculation of ferrets. The amniotic method of chick inoculation has given even poorer results than the inoculation of ferrets.

Quarterly Journal of Medicine, Oxford

10:139-282 (July) 1941

Bronchial Adenoma. A. F. Foster-Carter.—p. 139.
Refractory Anemia: I. Clinical and Pathologic Aspects. R. R. Bomford and C. P. Rhoads.—p. 175.
*Id.: II. Etiology and Treatment. R. R. Bomford and C. P. Rhoads.—p. 235.

Etiology of Refractory Anemia.—Bomford and Rhoads examined the records of 66 patients with refractory anemia in an attempt to evaluate the important etiologic factors. Race, sex, age and family history appeared to have no influence. A possible influence of eunuchism, menstruation and the menopause was noticed in a few instances. The incidence of achlorhydria was below normal. Half the patients were known to have been exposed to aromatic hydrocarbons and 1 to roentgen and radium therapy. Exposure to sunlight, ultraviolet and roentgen irradiation seemed to make the condition of a few patients worse. An increasing macrocytosis usually indicated a spontaneous remission. The results of tests of hepatic function suggested that hepatic impairment may be an important factor in the causation of refractory anemia. The simultaneous excretion of coproporphyrins I and III indicated that the disturbance of pigment metabolism in refractory anemia is similar to that observed in hepatic diseases and certain forms of exogenous poisoning but unlike that in pernicious anemia and hemolytic jaundice. The excretion of fecal urobilinogen was above normal levels in half the patients. The hemolytic substances normally present in human urine were present in a bound or conjugated form in the urine of patients with refractory anemia. The erythrocytes of these patients were slightly more than normally resistant to lysis by saponin. A possible abnormality in the processes of detoxication was detected. The measure of greatest importance in the treatment of patients with refractory anemia appeared to be the prevention of their exposure to any potentially hemotoxic substances or influences. Blood transfusions and dietary supplements were usually indicated for all patients, and splenectomy, as a last resort, for a few.

Jahresb. d. Kurashiki-Zentralhosp., Kurashiki

15:95-346 (July) 1941. Partial Index

*Studies on Japanese Type Epidemic Encephalitis: I. Histopathologic Study. Y. Kitagawa.—p. 95.
*Id.: II. Clinical Observation. Y. Kitagawa.—p. 145.

Japanese Type Encephalitis.—Kitagawa made histologic examination of the brain of 6 and the spinal cord of 5 patients who died of Japanese type epidemic encephalitis, with the following results: The predominating change in the brain in summer encephalitis is the increased accumulation of colloid cells, leading to the formation of colloid nodules and glia proliferation and resulting in the production of areas of softening. These nodules always arise at the point of entry of the virus in the parenchyma of the brain and bear definite relationship not only to the virulence of the virus but to individual constitution, as well as to the degree and extent of immunity. They appear to have special affinity for the midbrain, although they may be found throughout the central nervous system, particularly in the gray matter. These structures form primarily as a defensive process of the nervous system, but secondarily they may represent a means of disposing the necrotic material. The glial proliferation and entanglement may have the identical mechanism in their formation as the colloid nodules, and they may be regarded as a special form of Spielmeyer's pseudoneuronophages and true neuronophages. Microglia may show progressive changes in all parts of the nervous system, but the changes become stationary when the disease process ceases, while the changes in the oligodendroglia are usually negligible. Proliferation of macroglia practically never occurs in the acute stage of the disease. True neuronophages and perivascular infiltration are also of general distribution, but no special significance can be attached to these elements. Thus the summer encephalitis virus shows varying degrees of affinity to the central nervous system, producing changes in the nature of the blood. Primarily it stimulates the microglia, causing accumulation of colloid material at the port of entry and resulting in the formation of colloid nodules and glial proliferation; when it enters deeper into the parenchyma of the brain it causes changes and necrosis of the tissue. Although histologically summer encephalitis resembles von Economo's lethargic encephalitis, the author expresses the view that distinctive characteristics differentiate the nature and the distribution of the changes produced by these two diseases.

Clinical Observations of Japanese Encephalitis.—Kitagawa further reports his observations on the clinical manifestations of Japanese encephalitis in 26 typical patients, of whom 9 were male and 17 female, ranging in age from 10 to 56 years. The season of the year in which the disease most frequently occurred was the summer (July to September). The onset was acute, with cloudiness of the sensorium appearing within one to eight days, usually after the onset of high fever. The author describes in detail various neurologic signs of the disease, but special emphasis is placed on the state of the cerebrospinal fluid. In 26 cases a total of eighty-six examinations were made of the fluid obtained by lumbar puncture. The pressure varied from 45 to 300 mm., with an average of 196 mm. The reaction was always alkaline and the fluid, with only one exception in which slight turbidity was noted on one occasion, water clear. The globulin tests practically all gave positive results, but no tendency for pellicle formation was noted in any sample. The protein content of the eighty samples examined varied from 0.033 to 0.15 per cent, with an average of 0.075 per cent. The Heine sugar reaction was for the most part normal. The cell count in seventy-six samples revealed an average of 173 cells per cubic millimeter, the number decreasing with the progress of the disease. In 2 instances gram-positive diplococci were cultured. The hospital stay ranged from four to one hundred and seven days, the average being twenty-three days; the average duration of the disease with complete recovery was forty-five days. Relapse occurred in 4 cases, and the residual complications were elevation of temperature, disturbance of sleep, headache, nausea and vomiting and hypersecretion of saliva. Complications were as a rule rare, but empyema, pericarditis, pulmonary gangrene, pneumonia and bed sores occasionally followed the disease. The more serious after-effects were paralysis of the limbs, paralysis of the nerve and difficulty in deglutition. As a result of these observations the author concludes that the Japanese type of encephalitis is an entity distinct from the lethargic type, and that it is capable of terminating in parkinsonism.

Taiwan Igakkai Zassi, Taihoku, Formosa

40:1181-1348 (July) 1941. Partial Index

*Experiences with Chemotherapy in Gonorrheal and Nongonorrheal Urogenital Diseases. T. Yoh.—p. 1307.

Chemotherapy in Urologic Diseases.—Yoh employed sulfanilamide and its derivatives in a number of cases of urologic disease, including 140 of gonorrheal and 56 of nonspecific infections. Administration of massive doses of sulfanilamide in addition to the usual method of treatment definitely accelerated the cure of gonorrheal infection, but some of the complications resulting from gonorrhea failed to respond satisfactorily to this mode of treatment. If the patients with gonorrhea were first treated by the injection of either vaccine or autogenous blood, the subsequent employment of chemotherapy appeared to be more efficacious than it was when no preliminary treatment was instituted. The same beneficial effect was observed when treatment with sulfanilamide and its derivatives was preceded by fever therapy. It was difficult to differentiate the effect of the various sulfanilamide derivatives, but sulfapyridine appeared to be effective when other preparations failed to produce satisfactory results. Of the 32 patients who came to the attention of the author after a course of self medication with sulfanilamide or its derivatives only 1 was found to be cured; all the others still suffered from active disease. The drugs were also found to be effective in genitourinary diseases due to staphylococcic and streptococcic infections, but diseases due to the colon bacillus and the tubercle bacillus were resistant to chemotherapy. Sulfapyridine produced the greatest number of undesirable symptoms, but cessation of medication usually resulted in prompt disappearance of complicating manifestations. One instance of death due to granulocytopenia following massive doses of a sulfanilamide derivative is recorded.

Acta Medica Scandinavica, Stockholm

108:363-482 (Aug. 26) 1941. Partial Index

Multiple Myeloma with Leukemic Blood Picture and Degenerative Changes in Spinal Cord. G. A. Lindeboom and H. J. Mulder.—p. 363.
*Results of Specific Therapy of Leprosy in Estonia During Last Twenty Years. A. Paldrok.—p. 374.
Etiology of and Supposed Relations Between Lymphogranulomatosis Maligna and Mycosis Fungoides. O. Lövgren and C. Westman.—p. 387.
Clinical Studies on Chronic Articular Rheumatism. G. Edström.—p. 398.
*Some Cases of Macrocytic Hyperchromic Anemia Without Gastric Achylia, Their Etiology and Relation to Cryptogenic Pernicious Anemia and to a New Antianemic Factor. O. P. Nielsen.—p. 421.
Case of Hemochromatosis (Diabète Bronzé) Combined with Multiple Joint Anomalies. H. J. N. Dekkers.—p. 440.
Pellagra and Mental Diseases. H. Löfvendahl.—p. 455.
New Stomach-Liver Preparation for the Treatment of Pernicious Anemia. M. Odin.—p. 466.

Specific Therapy of Leprosy in Estonia.—Paldrok observed that the causal organism of leprosy consists of granules that are surrounded by an envelop; five or more are generally in one sheath. The common envelop creates the appearance of a rod which Amauer Hansen had designated as the bacillus of leprosy. Paldrok observed, however, that the multiplication of these rods is not like that of bacilli but that the granules adhere to the wall, then penetrate the envelop and develop into dendritic forms as do fungi. He became convinced that the causal organism of leprosy is not a bacillus but rather a fungus and that this explains why the leprosy organism does not grow in cultures suitable for bacilli. He found also that the granules and the covering differ in their chemical composition. Whereas the granules contain free nucleic acid, the envelop contains nucleoproteins. Searching for a substance that would destroy the covering so that the granules could be

reached, he decided to utilize the action of cold in the form of solid carbon dioxide. Rods of solid carbon dioxide are pressed against the leproma for three to four seconds. Lepromas of millet seed size completely disappear about three weeks after the freezing, but with larger ones the freezing must be repeated. At each session fifteen to twenty lepromas are frozen, and the procedure is repeated every two weeks. After four months, the treatment is interrupted for the same length of time. The decomposition products released during freezing are absorbed by the organism and cause the formation of antibodies. There are indications that the solid carbon dioxide treatment is an autoimmunization. The favorable effect of solid carbon dioxide on leprosy has been corroborated by investigators of many different countries. If after two years of treatment with solid carbon dioxide the organism has lost its responsiveness to the substance a new chemotherapeutic stimulus must be employed, and the author found the organic gold preparation solganal effective. In the course of treatment with solganal the organism regains its responsiveness to solid carbon dioxide, so that the two treatments can be used alternately. Good food, a hygienic mode of life, adequate exercise and weekly sweat baths are complementary measures. Those who are apparently cured are kept in the leprosarium for an additional two years. For the first five years after discharge the patients must submit to control examinations every six months. After pointing out that nonspecific shock therapy has largely failed in leprosy, the author says that the efficacy of the solid carbon dioxide and gold (solganal) treatment is proved by the fact that, whereas in 1920 the total number of patients with leprosy in Estonia was 316, by 1940 there were only 113.

Macrocytic Hyperchromic Anemia Without Gastric Achylia.—According to Nielsen it is now generally agreed that gestational pernicious anemia differs from cryptogenic pernicious anemia. Patients with the former often have free hydrochloric acid, have no organic nervous symptoms, seldom show an increased icterus index and do not require continuous liver therapy. In this report a brief survey is given of previous views concerning pernicious anemia with the gastric secretion of hydrochloric acid preserved and the pernicious anemia of pregnancy, with special reference to the cause and the relation to cryptogenic pernicious anemia. Histories are given of 2 cases of pernicious anemia of pregnancy and 2 cases of pernicious anemia with the gastric secretion of hydrochloric acid preserved. Therapeutically the diseases differ from cryptogenic pernicious anemia by their failure to respond to the injection of liver and stomach preparations. On the other hand, they respond favorably to oral administration of liver extract. The author advances the hypothesis that the cause is the same in the reported cases of pernicious anemia of pregnancy and pernicious anemia with preserved hydrochloric acid secretion, namely that the anemia is due to lack of a new antianemic factor. The diseases are taken to be essentially different from cryptogenic pernicious anemia but closely related to Israels and Wilkinson's achrestic anemia, and to tropical macrocytic anemia.

Acta Radiologica, Stockholm

22:439-534 (Sept. 15) 1941

Primary Cancer in Uterus and in Rectum of the Same Patient: Study of Twelve Case Histories. E. Nordlander.—p. 439.
Myelographic Changes in Kyphosis Dorsalis Juvenilis. E. Lindgren.—p. 461.
Primary Partial Invagination of Appendix Diagnosed by Roentgenologic Examination: Case. H.-G. Skarby.—p. 471.
*Significance of Large Niches in Stomach. H. Myhre.—p. 482.
Simple Method of Localizing Foreign Bodies. N. Westermark.—p. 490.
Terminal Ileitis. S. A. Chrom.—p. 493.
*Epituberculosis and Lung Atelectasis. N. Westermark.—p. 501.
Os Naviculare Bipartitum. E. Lindgren.—p. 511.
Darkrooms and Their Equipment in Roentgen Departments. N. Westermark.—p. 515.
*Results of Roentgen Treatment of Spondylosis Rhizomelica (Spondylarthritis Ankylopoietica). J. van Ebbenhorst Tengbergen and H. J. N. Dekkers.—p. 522.

Large Niches in Stomach.—According to Myhre a giant niche of the stomach always provokes a strong suspicion of cancer, because it is often stated that niches measuring more than 2.5 cm. in diameter are nearly always malignant. To throw more light on this problem, the author investigated 23 cases in which large niches had been photographed and in which operation was done. He included in this study all niches with an aperture of more than 2.5 cm., provided infiltration of the wall of the stomach outside the swollen edge of the niche did not clearly prove it to be a cancer. It was found that nine of the twenty-three craters were cancers, while the other fourteen were benign ulcers. Of the fourteen benign niches one measured 2.5 cm. in the roentgenogram, another 6.5 cm. and the others between 3 and 4.5 cm. Of the nine malignant ones one measured 2.5 cm. and the others between 3 and 4.5 cm. The surgically removed specimens usually had a smaller diameter than did the niche on the roentgenogram. Owing to divergence of rays, roentgenograms produce a magnification of 10 to 15 per cent. The author concludes that if no definite sign of cancer can be seen in the roentgenogram the large niches with an aperture of 3 cm. and more can be taken to represent benign ulcers at least as often as malignant ones, perhaps even more often. However, some really are malignant and the differential diagnosis may be impossible, because a roentgenologic improvement during dietetic treatment may prove misleading.

Epituberculosis and Pulmonary Atelectasis.—Westermark says that in 1920 Eliasberg and Neuland described a pathologic condition in tuberculous children which they designated epituberculosis. It is characterized by massive pulmonary consolidations. The roentgenologic aspects are similar to those of tuberculous pneumonia. Three different explanations have been given for the epituberculous opacity: 1. Tuberculous parenchymatous infiltrations are the cause of the opacity. 2. It is wholly or partly caused by obstructive atelectasis. 3. The opacity is caused by a focal reaction as a result of excessive sensitiveness. Reviewing the literature, the author found 44 cases of so-called epituberculosis and observed that among them two different types of changes could be differentiated. A group of 7 cases presents the aspects of a more or less extensive obstructive atelectasis, which is caused by enlarged hilar glands which infiltrate and compress the bronchus leading to the dense region. The opaque lobe occupies a smaller volume than it would normally. There is retraction of the thoracic wall over the opaque lobe and displacement of the heart and the mediastinum to the diseased side. The author describes a case of his own observation and reviews some cases from the literature. In the other condition, the opaque lobe is of normal or larger than normal volume; there is no retraction of the thoracic wall and no displacement of the heart and the mediastinum to the diseased side, but occasionally there is displacement to the healthy side. This process seems to tally with the true form of epituberculosis according to Eliasberg and Neuland. It is caused by a proliferative tuberculosis.

Roentgen Treatment of Rheumatoid Arthritis of Spine.—According to van Ebbenhorst Tengbergen and Dekkers roentgen treatment has been employed in rheumatoid arthritis of the spine (rhizomelic) for the last ten or twelve years. Since most investigators who tried this form of treatment obtained favorable results, particularly with regard to a reduction in pain, the authors likewise resorted to it. In the 27 patients on whom they employed roentgen treatment the diagnosis was well established. The majority had had their complaint for a long time, because the diagnosis is often made rather late. Before roentgen treatment was begun some form of physical therapy had been used on all patients. Some had received injections of the Warren-Crowe vaccine, mostly without much beneficial effect. The authors outline the technical details of the roentgen therapy. The best effects were obtained in the patients in whom the symptoms had existed for only a comparatively short time (less than one year). Summarizing the results of therapy the authors state that 16 patients showed considerable and 2 moderate subjective improvement; 15 were able to continue their daily work. The function of the spinal column was improved in only 4 of the patients. The sedimentation rate decreased in 18 cases. If the sedimentation speed is regarded as an index of the activity of the process, it may be said that in 18 cases the activity was subdued. The authors think that on the whole the results may be designated as favorable.

Book Notices

The Modern Treatment of Syphilis. By Joseph Earle Moore, M.D., Associate Professor of Medicine, The Johns Hopkins University, Baltimore. With the collaboration of Jarold E. Kemp, M.D., Associate in Venereal Diseases, The Johns Hopkins University, et al. Second edition. Cloth. Price, $7. Pp. 674, with 98 illustrations. Springfield, Illinois & Baltimore: Charles C. Thomas, 1941.

There are many changes in this edition. As the preface states, twenty-three of the thirty-two chapters of the first edition have been completely rewritten. Moreover, the material on infectiousness in syphilis has been greatly amplified, particularly in its relation to treatment. There is a new chapter on public health aspects of syphilis and a much needed chapter devoted to the subject of intensive arsenotherapy. The chapter on interpretation of serologic tests in the diagnosis and treatment of syphilis has been completely rewritten by Dr. Harry Eagle and adds much to the value of the book. Dr. Paul Padget has revised the chapter on cardiovascular syphilis and Dr. Mary Goodwin has completely revised the chapters on the treatment of the syphilitic pregnant woman and on the antepartum prevention of congenital syphilis, as well as on the treatment of congenital syphilis. As Dr. Moore well puts it, Dr. Jarold Kemp, only recently deceased, a clinician, immunologist and syphilologist of the highest ability, assisted greatly in the complete revision of the entire manuscript.

The reviewer has noted only a few minor items to which exception might be taken. On page 136 the advice is given that biliposol, a liposoluble compound, be given two or three times a week. It is true that on page 141 its use is more properly advised every five to seven days. Again, thiobismol is recommended as an antisyphilitic injection twice weekly. It should be administered more properly three times a week, and because of its rapid excretion there is a question whether this drug should ever be used as an antisyphilitic agent. On page 190, last paragraph, the word "gland" is improperly employed for "lymph node." On the same page the use of B. hemophilus Ducrey vaccine for the treatment of chancroidal infection is not even mentioned, yet it is very useful for syphilitic patients getting arsenical therapy when one might hesitate to use sulfonamide derivatives internally.

Moore feels, page 247, that, for syphilitic patients undergoing treatment with bismuth and arsenic compounds examination of the urine once in two to three months is sufficient. The reviewer thinks that a more conservative period would be once a month. On pages 269 and 278, in late latent syphilis and in benign late syphilis, osseous and mucocutaneous, the advice is given to start therapy with an arsenical. Would it not be better to institute first a course of bismuth compounds? After all, modern methods of diagnosis are not entirely able to detect certain submerged syphilitic changes in the aorta and liver. The reviewer thinks that the first lumbar puncture should be done earlier in the course of the disease rather than at six months. There is difference of opinion, however (page 349). Otherwise, certain persons with early central nervous system involvement will not have their lesions detected.

On page 386, figure 72, the word "thiobismol" is misspelled and the addendum should be "from Cole, Schwartz et al."

But these are only minor items in what is an admirable presentation of the treatment of syphilis. The entire subject is so well handled that it is difficult to pick out exceptional chapters, though the portions on infectiousness, on central nervous system syphilis and on interpretation of serologic tests are particularly well handled. The book will be a "required" standard for every hospital, syphilis clinic and physician and student of syphilis. The authors are to be congratulated for their comprehensive treatment of such an important and almost endless branch of medicine.

The volume is replete with tables and charts to illustrate better the subjects under discussion. Moreover, the printer and the bookbinder have done their work well.

What a pity it is that Kemp is not here to enjoy the deserved encomiums due its authors.

El factor emocional en la etiopatogenia de la enfermedad de Basedow. El mecanismo causal analizado en 24 casos: Conclusiones, patogenicas, terapeuticas, pronosticas y profilacticas. Por el Dr. Leonardo C. Perrusi, adscripto a la Cátedra de clinica médica de la Facultad de ciencias, médicas de Buenos Aires. Paper. Pp. 190. Buenos Aires: "El Ateneo," 1939.

In this book the author has made careful and detailed psychoanalytic studies of a group of 24 selected cases of hyperthyroidism and in each instance he finds some psychic trauma responsible for the initiation of the disease. For example, each chapter contains the report of a case of hyperthyroidism, and the titles run as follows:

Chapter II. Basedow's Disease of Psychogenic Origin, Brought About by a Sudden Fright.
Chapter III. Basedow's Disease Aroused by an Inferiority Complex and by Exaggerated Ambitions and Libido.
Chapter IV. Psychogenic Basedow's Provoked by an Insolvable Family Situation.
Chapter V. Basedow's Provoked by Unmanagreable Pruritus.
Chapter VI. Basedow's Orginated by a Sentimental Drama.
Chapter VII. Basedow's Disease Brought About by Antivenereal Phobia.
Chaper VIII. Basedow's Disease Brought About by the Death of a Relative and a Situation of Desperation.
Chapter IX. Basedow's Brought About by the Prolonged Sickness of the Husband.

After analyzing these and 15 other cases, the author concludes that in almost all cases of hyperthyroidism the disease is produced by emotional factors which excite the gland to hyperactivity via the sympathetic nervous system.

The author feels critical of those physicians who make a diagnosis of hyperthyroidism of unknown origin and states that this diagnosis is incorrect, as the majority of the cases that the author has seen could be classified as dysthyroidism occurring in predisposed persons as a result of emotional disturbances.

The author admits that the factors influencing the predisposition of a patient to acquire hyperthyroidism are poorly understood. Among predisposing factors he lists the degree of civilization, the activity of the patient's life, the geographic position of the country, emotional disturbances, inferiority complexes, conception during a difficult period in the parent's life, and inharmonious function of the endocrine glands.

The author emphasizes that, in addition to the usual treatment of hyperthyroidism by rest, iodine, roentgen rays or surgery, all patients with hyperthyroidism should be subjected to psychoanalytic studies to determine and eradicate the emotional factors responsible for the development of the disease.

There are few who would agree with the author's conclusions as to the etiology of hyperthyroidism. The trend of thought is away from the conception that it is the result of worry, anxiety, fear or any other emotional disturbance. Few would deny that emotional disturbances result in an exacerbation of the symptoms of a patient already suffering from hyperthyroidism, but even fewer would subscribe to the hypothesis that the disease is caused by emotional disturbances. Dr. Perrusi fails to present any new evidence to support the concept of an emotional etiology of hyperthyroidism, nor does he present any logical reasoning in support of such a theory.

His cases, although obviously carefully studied from the psychoanalytic point of view, are not always convincing, and the emotional factors which the author assumes initiated the hyperthyroidism are often far fetched. It would be hard indeed to find any series of 24 cases of any disease in which, if one looked deeply enough into the emotional background of the patient, one could not find emotional factors as significant as those reported in the author's cases.

The book is interesting and carefully worked out from the psychoanalytic standpoint but unconvincing.

Hypoparathyroidism in Denmark: A Clinical Study. By Aage Lachmann. Translated from Danish by Hans Andersen, M.D. Acta medica Scandinavica Supplementum CXXI. Paper. Pp. 260, with 14 illustrations. Copenhagen: Einar Munksgaard, 1941.

Acta medica Scandinavica contains articles pertaining to internal medicine. These articles are published in English, French or German as the authors prefer. The present volume, a clinical study of hypoparathyroidism in Denmark, is printed in English with a summary in Danish and represents work which was begun in 1934. The material has been divided into an introduction, discourses on postoperative hypoparathyroidism, idiopathic hypoparathyroidism, a summary and case records. A

[illegible]

[illegible]

This is an interesting and instructive presentation of [illegible] cases of [illegible]. [illegible]

A insuficiência coronária (estudo anatomoclínico). [illegible]

It is gratifying to have so lucid a review of coronary insufficiency printed in Portuguese. The author has thoroughly reviewed the literature, and a perusal of the monograph shows that he is cognizant of the recent developments in this field. The bibliography of one hundred and sixty-three references covers the American, South American and European literature adequately. The subject is divided into five sections. The first deals with the anatomy and physiology of the coronary vessels, the second with the general concept of coronary insufficiency, the third with its causes and the mechanisms responsible for it, the fourth with the clinical manifestations and the last with the electrocardiographic appearance. The author includes a series of his own cases in the last section. There is little that the physicians of this country can obtain from this monograph not present in excellent reviews on this subject in the English literature.

Alimentación y nutrición en Colombia. Por el Dr. Jorge Bejarano, profesor de higiene de la Facultad de medicina de Bogotá. Paper. Pp. 219. Bogotá: Editorial Cromos, 1941.

This is a good book written by a man who is professor of hygiene at the University of Bogota and president of the Red Cross of Colombia. Dr. Bejarano is concerned over the lack of good judgment in the choice of foods which he finds among so many of the people of Colombia. They take too much sugar and starchy food and do not get enough protein and fat. They take little milk and eggs, and they do not get enough fruit and vegetables. Many persons imbibe altogether too much of the [illegible] In some places the water supplies are poorly protected from contamination with fecal material. Dr. Bejarano has written a book which will give Spanish speaking physicians and intelligent laymen a good idea of the recent advances in the science of alimentation and nutrition. He hopes that more of this knowledge can be put to use in his country.

[illegible]

The author reviews the observations made in a series of [illegible] patients seen in the obstetric and gynecologic clinic [illegible] of the Medical School of Buenos Aires. [illegible] He states that, in relief of pain, success was obtained in [illegible] per cent of cases, that relative failure was obtained in [illegible] per cent and that failure resulted in [illegible] per cent of cases. No untoward effect was observed with regard to the mother or the infant or in the progress of labor in [illegible] per cent of cases, whereas in [illegible] per cent complications arose. The author believes that, on an average, complete success was obtained in [illegible] per cent of cases. He believes that the drugs which he describes should be used in the first stage of labor but that during the actual birth of the child gas anesthesia or regional anesthesia should be used and that the physician should not use one method of anesthesia as a matter of routine.

Die Nervenkrankheiten des Rindes: Eine Grundlage für eine vergleichende Neurologie von Mensch und Haustier. Von Dr. med. E. Frauchiger und Dr. med. vet. W. Hofmann, [illegible]. Pp. 381, with [illegible] illustrations. Bern: Hans Huber, 1941.

This book on the nervous diseases of cattle and live stock is a real contribution. It is a groundwork for a comparative neurologic study of man and animal. The majority of the illustrations are in relation to cattle. There are two parts to this excellent book. The first part consists of a short abstract of the nervous diseases of the animals together with the clinical anatomy and methods of study. The second portion consists of the diseases of the brain, circumscribed disorders of the brain, diseases of the spinal cord, lesions of the cortices of the central nervous system, peripheral nerve diseases, disturbances of the vegetative nervous system, disorders of the muscles and, lastly, diseases of the nervous system without organic disease (neuroses). There is a good bibliography after each chapter in the two portions of the book. The German is easily read and the statements are clearly written. This book is a most unusual one and is highly recommended to all neuropathologists, neurologists and veterinarians.

Synopsis of Applied Pathological Chemistry. By Jerome E. Andes, M.S., Ph.D., M.D., Director of Department of Health and Medical Advice, University of Arizona, Tucson, and A. G. Eaton, B.S., M.A., Ph.D. Assistant Professor of Physiology, Louisiana State University School of Medicine, New Orleans. Fabrikoid. Price, $4. Pp. 429, with 23 illustrations. St. Louis: C. V. Mosby Company, 1941.

In this handy, pocket sized volume are gathered practically all of the well established chemical methods applicable to the study of blood, cerebrospinal fluid and urine. In addition, chapters are devoted to renal, liver and endocrine functional tests and to gastric analysis and basal metabolism. The physiologic and pathologic changes in concentration of the various substances considered are discussed and well charted in relation to clinical findings. Concise statements of procedure, supplemented by references to adequate but less well known methods, render the book of practical value to the intern and the busy physician. The book is not without some minor inaccuracies of statement, and it can hardly be classified as a reference work. In the main, however, it admirably fulfils the authors' purpose by providing a "practical, simple, easily read text on the application of pathologic chemistry to clinical medicine."

Queries and Minor Notes

THE ANSWERS HERE PUBLISHED HAVE BEEN PREPARED BY COMPETENT AUTHORITIES. THEY DO NOT, HOWEVER, REPRESENT THE OPINIONS OF ANY OFFICIAL BODIES UNLESS SPECIFICALLY STATED IN THE REPLY. ANONYMOUS COMMUNICATIONS AND QUERIES ON POSTAL CARDS WILL NOT BE NOTICED. EVERY LETTER MUST CONTAIN THE WRITER'S NAME AND ADDRESS, BUT THESE WILL BE OMITTED ON REQUEST.

P WAVE AND CORONARY DISEASE

To the Editor:—A man aged 51 had an electrocardiogram made during the course of a general examination, on Dec. 30, 1940. At that time he gave no history or symptoms of heart disease, but the report of the cardiologist was as follows: "Electrocardiogram essentially normal. The negative P waves in leads 2 and 3 are not believed to be of clinical significance." About the middle of July 1941 the man began to complain of such symptoms as dyspnea on exertion and substernal pain on climbing stairs. About the first of August he became ill with an acute bronchitis, and since that time he has noted heart consciousness, dyspnea on exertion and chronic nonproductive cough especially after retiring at night. He has not had typical anginal pain radiating from the precordium or up into the neck. An electrocardiogram report on Sept. 26, 1941 states that "P wave changes, including widening, suggest auricular disease—may be functional." The cardiologist further states that "In view of the history and evidence of peripheral arteriosclerosis as shown by moderate thickening of the peripheral arteries and as suggested by electrocardiographic findings, coronary arteriosclerosis is considered to be present. No evidence of cerebral arteriosclerosis (eyegrounds essentially normal)." Another electrocardiogram was made on Oct. 23, 1941 by a different physician, and while the cardiogram appears the same as the one made in September, he states that it is "not definitely abnormal." It is asked that significance of inverted or negative P waves in leads 2 and 3 be explained. Could symptoms as mentioned above in a patient had left a quiet occupation for one requiring rather strenuous exertion be a causative factor in producing a coronary sclerosis? In other words, would the cardiogram taken in conjunction with subjective symptoms and with negative findings on physical examination of the heart be indicative of beginning coronary disease? M.D., Arkansas.

ANSWER.—Inversion of P in lead 3 occurs normally, but P inversion in lead 2 is abnormal. The best known changes in the P wave are the broad, tall, notched and upright P waves in leads 1 and 2 which occur in mitral stenosis, and the tall upright P waves in leads 2 and 3 in chronic cor pulmonale. Inversion of P_2 and P_3 usually indicate a change in the direction of spread of the impulse in the auricles. The most common cause is retrograde stimulation of the auricles by a pacemaker located in the auriculoventricular node. This is accompanied by a shortened PR interval (less than 0.12 second). If the PR is not shortened, it is presumed that a sinus rhythm is associated with intra-auricular conduction disturbance. The significance of an inverted P_2 and P_3 depends on the condition giving rise to activity of the subsidiary nodal pacemaker when this is the mechanism of its production or to the cause of the intra-auricular conduction disturbance when the pacemaker is still in the sinus node. Transient P wave inversion is less significant than permanent P wave inversion.

Coronary disease is one of the common causes for P wave inversion in leads 2 and 3, although infectious diseases or excessive digitalis may be responsible in part. Other evidences of abnormalities in the electrocardiogram will help evaluate the meaning of P wave inversion. In short, inversion of P_2 and P_3 is abnormal, but its true meaning must depend on an analysis of the contour of the P wave and its duration, the PR duration, the contour of the ventricular complex and the rate of the heart.

When none of these other factors are altered, too much attention should not be attached to such P wave inversions.

As regards exertion as a causative factor in producing coronary sclerosis, there is no evidence to favor this view other than a philosophical belief that "burning the candle at both ends" leads to more rapid aging of the coronary arteries. It seems more likely in this case to assume that with a more strenuous life the signs of coronary insufficiency become more manifest. The presence of the symptoms in this case are far more significant than the P wave abnormalities, and only by a proper evaluation at the bedside of the circumstances which might give rise to these symptoms can the final judgment be made as to whether or not clinically significant coronary disease is present.

NIH SWABS FOR PINWORMS

To the Editor:—Can you give me some information on the NIH cellophane swab frequently mentioned in connection with pinworms?

M.D., Illinois.

ANSWER.—The NIH cellophane swab was named by the late Dr. Maurice C. Hall, former chief of the Division of Zoology of the National Institute of Health; hence the initials NIH. Hall had found that only approximately 5 per cent of all the cases of oxyuriasis can be diagnosed from fecal examination. He found from the literature that various types of swabbings and scrapings made from the perianal area provided a much better method of diagnosis. He perfected a simple swab consisting of a glass rod about $\frac{7}{16}$ inch in diameter and 4 inches in length and with a small square or circle of cellophane attached to one end with a small rubber band. The swabbing is made preferably in the morning on the suspected patient before the patient has had his morning stool and before he takes a bath. The skin of the perianal and perineal region is swabbed with the cellophane, and if this is at a home call on the patient it is then placed in a stoppered test tube and taken to the office or clinical laboratory. The cellophane is then removed with a pair of curved forceps, flattened out on a slide in a few drops of distilled water and mounted with a coverglass. Eggs of the seatworm, which will have readily adhered to the cellophane, are therefore easily recognized in the transparent preparation. In order to be absolutely sure that the patient does or does not have oxyuriasis, it is necessary to make a series up to six swabbings, unless, of course, the eggs are previously found. Furthermore, since oxyuriasis is more or less familial, if two or more members of the family are diagnosed as infected it is usually advisable to treat the whole family, including the father.

STRABISMUS IN YOUNG CHILD

To the Editor:—Nine weeks ago a boy aged 2 years fell from the steps and struck his head on concrete, landing on the superior lateral portion of the left orbit. He did not become unconscious, and the injury seemed so trivial that a physician was not called. Three weeks later the child went to bed apparently perfectly normal and awakened in the morning with a decided internal squint of the left eye, which to date has shown no signs of improvement. I was consulted on the day the squint occurred. I delivered this child, and the delivery was perfectly normal. The boy's head was roentgenographed, and there were no signs of fracture, although the physician who made the roentgenogram stated that roentgenograms taken at this age frequently are unsatisfactory. I referred the parents to an eye specialist, who believes the condition to be paralysis of the abducens due to the injury, possibly resulting from the formation of a small hematoma, and also states that he feels sure it is not a Gradenigo syndrome. I referred them also to a local neurologist, who states that he believes it to be not the result of an injury but paralysis of the abducens, cause unknown, a condition which occurs in children between the ages of about 2 to 4 years but which may but does not always clear up around the age of 14. I would appreciate information on similar case records of spontaneous or traumatic paralysis of the abducens. Assuming that this is paralysis of the abducens without remission, is there any operation on the eye muscles which will relieve the condition, and if so at what age should this be done so that the sight of the eye will not be impaired? Do you have any other studies to suggest or any treatment? M.D., Pennsylvania.

ANSWER.—It is impossible to tell in the absence of details of the examination whether the condition is a paralysis or a concomitant strabismus. The injury is an unlikely cause owing to the long interval between it and the onset of the squint (three weeks, in a very young child). It is a common history that strabismus in a child begins abruptly, the early stages being overlooked by the family. Paralysis of the abducens dating from birth is not uncommon. It is decidedly uncommon coming on spontaneously at the age of 2 years. Convergent strabismus coming on at this age is exceedingly common. Paralysis produces diplopia, which might cause the child to hold his head in a position to avoid diplopia (turned toward the right). In paralysis, if the patient is made to fixate first with the normal eye and then with the paralyzed eye, the other eye being covered, the deviation of the normal eye under cover, when the paralyzed eye fixates, is greater than the deviation of the paralyzed eye when the normal eye fixates. In a concomitant strabismus the deviation is approximately equal. Cases of true paralysis tend to become concomitant before long.

In the absence of more data a diagnosis cannot be made. The prognosis for spontaneous recovery is not good. This is true whether the condition is paralytic or not. Care should be taken that the squinting eye does not become amblyopic from disuse (suppression to avoid diplopia). Should signs of this appear, the good eye must be occluded to compel use of the other eye. This is important for several years. If due care is exercised along these lines, operation may be postponed indefinitely without the sight becoming impaired. Operation to straighten the eye gives good results and should be done before the patient acquires an inferiority complex due to consciousness of his defect. The operative treatment is satisfactory. The operation of choice depends on whether there is a total paralysis or a moderate or a slight paresis and should of course be determined by the surgeon to whom the operation is entrusted. Often more than one operation is needed.

bibliography of three hundred and forty-eight references is included and should be of inestimable value to those interested in the clinical study of hypoparathyroidism. The format is conducive to easy reading, and the descriptions follow in well ordered sequence.

Of special interest is the author's review of the individual symptoms in postoperative hypoparathyroidism. He has divided them into three groups: (1) changes in calcium and phosphorus metabolism, (2) symptoms from the nervous system and (3) trophic changes in the ectodermal tissues. In the study of agents capable of raising the serum calcium level, particular attention is paid to calcium preparations, vitamin D preparations, acidifying remedies, parathyroid hormones, parathyroid transplantations and combinations of the foregoing. Dihydrotachysterol gave the most promising results. For those who are interested in the problem of hypoparathyroidism, this treatise can with little reservation be recommended for correlative reading.

Orbital Tumors: Results Following the Transcranial Operative Attack. By Walter E. Dandy. Cloth. Price, $5. Pp. 168, with 100 illustrations. New York: Oskar Piest, 1941.

This is an interesting and instructive presentation of 31 cases of tumors involving the orbit. The 24 cases in which the orbital portion of the tumor was attacked surgically by the transcranial approach again give ample testimony to the surgical skill of the author. In addition to the detailed presentation of the cases there is a discussion of the pathologic nature of the tumors encountered and of the operative procedure. With such wide variations in the microscopic appearance of the lesions described here as Christian-Schüller's disease, one shares the author's reservations regarding the diagnosis of these cases. It is not a little surprising to find the common meningioma referred to here as a "dural endothelioma," a "dural tumor" or a "dural meningioma" when practically every modern pathologist with any considerable experience with these tumors has come to recognize their origin from the leptomeninx. It is rather surprising that two procedures, insertion of a flanged needle into the lateral ventricle for continuous drainage throughout the operation (in addition to opening of the chiasmatic cistern) and the use of motor driven burrs for the removal of markedly thickened bone, which other surgeons have found of great value, have apparently found no place among the surgical procedures of this author. The illustrations, particularly the drawings illustrative of the operative procedures, are excellent and add much to the value of this monograph. Ophthalmologists and neurosurgeons alike will wish to add this book to their libraries.

A insuficiencia coronária (estudo semiológico). Por Caio Benjamin Dias, assistente de Clínica propedêutica médica da Faculdade de medicina da U. M. G. Paper. Pp. 162, with 51 illustrations. Belo Horizonte: Livraria Editora Paulo Bluhm, 1941.

It is gratifying to have so lucid a review of coronary insufficiency printed in Portuguese. The author has thoroughly reviewed the literature, and a perusal of the monograph shows that he is cognizant of the recent developments in this field. The bibliography of one hundred and sixty-three references covers the American, South American and European literature adequately. The subject is divided into five sections. The first deals with the anatomy and physiology of the coronary vessels, the second with the general concept of coronary insufficiency, the third with its causes and the mechanisms responsible for it, the fourth with the clinical manifestations and the last with the electrocardiographic appearance. The author includes a series of his own cases in the last section. There is little that the physicians of this country can obtain from this monograph not present in excellent reviews on this subject in the English literature.

Alimentación y nutrición en Colombia. Por el Dr. Jorge Bejarano, profesor de higiene de la Facultad de medicina de Bogotá. Paper. Pp. 170. Bogotá: Editorial Cromos, 1941.

This is a good book written by a man who is professor of hygiene at the University of Bogota and president of the Red Cross of Colombia. Dr. Bejarano is concerned over the lack of good judgment in the choice of foods which he finds among so many of the people of Colombia. They take to much sugar and starchy food and do not get enough protein and fat. They take little milk and eggs, and they do not get enough fruit and vegetables. Many persons imbibe altogether too much of the locally made alcoholic drinks, and they fail to get enough calories in the diet. In most places the water supplies are poorly guarded from contamination with fecal material. Dr. Bejarano has written a book which will give Spanish speaking physicians and intelligent laymen a good idea of the recent advances in the science of alimentation and nutrition. He hopes that more of this knowledge can be put to use in his country.

Analgesia obstétrica: El sueño crepuscular barbitúrico en el parto. Con estudio especial del pentobarbital sódico. Por Juan Leon, profesor adjunto de Clínica obstétrica de la Facultad de ciencias médicas de Buenos Aires. Paper. Pp. 374, with 169 illustrations. Buenos Aires: El Ateneo, 1941.

The author reviews the observations made in a series of 201 patients seen in the obstetric and gynecologic clinic Elisio Cantón of the Medical School of Buenos Aires. In this series pentobarbital sodium was used alone in 80 cases, pentobarbital sodium and scopolamine were used in 40 cases, pentobarbital sodium and paraldehyde were used in 40 cases and pentobarbital sodium and chloral hydrate were used in 50 cases. Leon discusses the effects of these drugs from the standpoint of relief of pain, their effect in shortening or increasing the length of labor, their freedom from aggravation of infections and their effect on the child as far as respiration is concerned. He states that, in relief of pain, success was obtained in 39.6 per cent of cases, that relative failure was obtained in 49 per cent and that failure resulted in 9.5 per cent of cases. No untoward effect was observed with regard to the mother or the infant or in the progress of labor in 76.1 per cent of cases, whereas in 23.9 per cent complications arose. The author believes that, on an average, complete success was obtained in 30.5 per cent of cases. He believes that the drugs which he describes should be used in the first stage of labor but that during the actual birth of the child gas anesthesia or regional anesthesia should be used and that the physician should not use one method of anesthesia as a matter of routine.

Die Nervenkrankheiten des Rindes: Eine Grundlage für eine vergleichende Neurologie von Mensch und Haustier. Von Dr. med. E. Frauchiger und Dr. med. vet. W. Hofmann, ord. Prof. für Rinderheilkunde an der Universität Bern. Cloth. Price, 24 Swiss francs. Pp. 361, with 139 illustrations. Berne: Hans Huber, 1941.

This book on the nervous diseases of cattle and live stock is a real contribution. It is a groundwork for a comparative neurologic study of man and animal. The majority of the illustrations are in relation to cattle. There are two parts to this excellent book. The first part consists of a short abstract of the nervous diseases of the animals together with the clinical anatomy and methods of study. The second portion consists of the diseases of the brain, circumscribed disorders of the brain, diseases of the spinal cord, lesions of the cortices of the central nervous system, peripheral nerve diseases, disturbances of the vegetative nervous system, disorders of the muscles and, lastly, diseases of the nervous system without organic disease (neuroses). There is a good bibliography after each chapter in the two portions of the book. The German is easily read and the statements are clearly written. This book is a most unusual one and is highly recommended to all neuropathologists, neurologists and veterinarians.

Synopsis of Applied Pathological Chemistry. By Jerome E. Andes, M.S., Ph.D., M.D., Director of Department of Health and Medical Advisor, University of Arizona, Tucson, and A. G. Eaton, B.S., M.A., Ph.D. Assistant Professor of Physiology, Louisiana State University School of Medicine, New Orleans. Fabrikoid. Price, $4. Pp. 428, with 23 illustrations. St. Louis: C. V. Mosby Company, 1941.

In this handy, pocket sized volume are gathered practically all of the well established chemical methods applicable to the study of blood, cerebrospinal fluid and urine. In addition, chapters are devoted to renal, liver and endocrine functional tests and to gastric analysis and basal metabolism. The physiologic and pathologic changes in concentration of the various substances considered are discussed and well charted in relation to clinical findings. Concise statements of procedure, supplemented by references to adequate but less well known methods, render the book of practical value to the intern and the busy physician. The book is not without some minor inaccuracies of statement, and it can hardly be classified as a reference work. In the main, however, it admirably fulfils the authors' purpose by providing a "practical, simple, easily read text on the application of pathologic chemistry to clinical medicine."

Queries and Minor Notes

THE ANSWERS HERE PUBLISHED HAVE BEEN PREPARED BY COMPETENT AUTHORITIES. THEY DO NOT, HOWEVER, REPRESENT THE OPINIONS OF ANY OFFICIAL BODIES UNLESS SPECIFICALLY STATED IN THE REPLY. ANONYMOUS COMMUNICATIONS AND QUERIES ON POSTAL CARDS WILL NOT BE NOTICED. EVERY LETTER MUST CONTAIN THE WRITER'S NAME AND ADDRESS, BUT THESE WILL BE OMITTED ON REQUEST.

P WAVE AND CORONARY DISEASE

To the Editor:—A man aged 51 had an electrocardiogram made during the course of a general examination, on Dec. 30, 1940. At that time he gave no history or symptoms of heart disease, but the report of the cardiologist was as follows: "Electrocardiogram essentially normal. The negative P waves in leads 2 and 3 are not believed to be of clinical significance." About the middle of July 1941 the man began to complain of such symptoms as dyspnea on exertion and substernal pain on climbing stairs. About the first of August he became ill with an acute bronchitis, and since that time he has noted heart consciousness, dyspnea on exertion and chronic nonproductive cough especially after retiring at night. He has not had typical anginal pain radiating from the arms or up into the neck. An electrocardiogram report on Sept. 26, 1941 states that "P wave changes, including widening, suggest auricular disease—may be functional." The cardiologist further states that "In view of the history and evidence of peripheral arteriosclerosis as shown by moderate thickening of the peripheral arteries and as suggested by electrocardiographic findings, coronary arteriosclerosis is considered to be present. No evidence of cerebral arteriosclerosis (eyegrounds essentially normal)." Another electrocardiogram was made on Oct. 23, 1941 by a different physician, and while the cardiogram appears the same as the one made in September, he states that it is "not definitely abnormal." It is asked that significance of inverted or negative P waves in leads 2 and 3 be explained. Could symptoms as mentioned coming on after the patient had left a quiet occupation for one requiring rather strenuous exertion be a causative factor in producing a coronary sclerosis? In other words, would the cardiogram taken in conjunction with subjective symptoms and with negative findings on physical examination of the heart be indicative of beginning coronary disease? M.D., Arkansas.

ANSWER.—Inversion of P in lead 3 occurs normally, but P inversion in lead 2 is abnormal. The best known changes in the P wave are the broad, tall, notched and upright P waves in leads 1 and 2 which occur in mitral stenosis, and the tall upright P waves in leads 2 and 3 in chronic cor pulmonale. Inversion of P_1 and P_2 usually indicate a change in the direction of spread of the impulse in the auricles. The most common cause is retrograde stimulation of the auricles by a pacemaker located in the auriculoventricular node. This is accompanied by a shortened PR interval (less than 0.12 second). If the PR is not shortened, it is presumed that a sinus rhythm is associated with intra-auricular conduction disturbance. The significance of an inverted P_2 and P_3 depends on the condition giving rise to activity of the subsidiary nodal pacemaker when this is the mechanism of its production or to the cause of the intra-auricular conduction disturbance when the pacemaker is still in the sinus node. Transient P wave inversion is less significant than permanent P wave inversion.

Coronary disease is one of the common causes for P wave inversion in leads 2 and 3, although infectious diseases or excessive digitalis may be responsible in part. Other evidences of abnormalities in the electrocardiogram will help evaluate the meaning of P wave inversion. In short, inversion of P_2 and P_3 is abnormal, but its true meaning must depend on an analysis of the contour of the P wave and its duration, the PR duration, the contour of the ventricular complex and the rate of the heart.

When none of these other factors are altered, too much attention should not be attached to such P wave inversions.

As regards exertion as a causative factor in producing coronary sclerosis, there is no evidence to favor this view other than a philosophical belief that "burning the candle at both ends" leads to more rapid aging of the coronary arteries. It seems more likely in this case to assume that with a more strenuous life the signs of coronary insufficiency become more manifest. The presence of the symptoms in this case are far more significant than the P wave abnormalities, and only by a proper evaluation at the bedside of the circumstances which might give rise to these symptoms can the final judgment be made as to whether or not clinically significant coronary disease is present.

NIH SWABS FOR PINWORMS

To the Editor:—Can you give me some information on the NIH cellophane swab frequently mentioned in connection with pinworms? M.D., Illinois.

ANSWER.—The NIH cellophane swab was named by the late Dr. Maurice C. Hall, former chief of the Division of Zoology of the National Institute of Health; hence the initials NIH. Hall found that only approximately 5 per cent of all the cases of oxyuriasis can be diagnosed from fecal examination. He found from the literature that various types of swabbings and scrapings made from the perianal area provided a much better method of diagnosis. He perfected a simple swab consisting of a glass rod about $\frac{3}{16}$ inch in diameter and 4 inches in length and with a small square or circle of cellophane attached to one end with a small rubber band. The swabbing is made preferably in the morning on the suspected patient before the patient has had his morning stool and before he takes a bath. The skin of the perianal and perineal region is swabbed with the cellophane, and if this is at a home call on the patient it is then placed in a stoppered test tube and taken to the office or clinical laboratory. The cellophane is then removed with a pair of curved forceps, flattened out on a slide in a few drops of distilled water and mounted with a coverglass. Eggs of the seatworm, which will have readily adhered to the cellophane, are therefore easily recognized in the transparent preparation. In order to be absolutely sure that the patient does or does not have oxyuriasis, it is necessary to make a series up to six swabbings, unless, of course, the eggs are previously found. Furthermore, since oxyuriasis is more or less familial, if two or more members of the family are diagnosed as infected it is usually advisable to treat the whole family, including the father.

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CHEMICAL DISINFECTION OF INSTRUMENTS

To the Editor:—I am interested in using anhydrous acetone as a sterilizing agent for syringes, knives and instruments. I find that when I use a germicide containing formaldehyde I must rinse the syringes in distilled water before using them. I have been told that acetone can be used instead and that syringes may be taken out of the solution, air dried and used for intravenous work. After lancing pus pockets I usually soak the knives in 70 per cent rubbing alcohol for one hour. Could they then be returned to the acetone solution and used later without further sterilization? Please advise as to whether or not acetone is a satisfactory bactericidal agent. I have also been told that 99 per cent isopropyl alcohol is satisfactory for this purpose, but I find that it takes too long for air drying and leaves a greasy film on the syringe. A chemist suggested to me that a mixture of two thirds acetone and one third isopropyl alcohol would also be suitable for this purpose. Do you know of any other type of anhydrous antiseptic or bactericidal agent which would not rust instruments because of their water content and yet would still sterilize, and from which syringes could be air dried and used for intravenous work? If so would these agents be superior to the acetone or isopropyl alcohol or a mixture of the two?

M.D., New Jersey.

Answer.—Chemical disinfection of instruments is a problem which has not yet been solved satisfactorily. Spaulding (*Surg., Gynec. & Obst.* **69**:738 [Dec.] 1939) tested seven solutions commonly used for "cold sterilization" of instruments and found that, although these agents were able to kill vegetative bacteria within ten minutes, eighteen hours or longer was required by all of them to destroy sporulating organisms. The best results were obtained with formaldehyde-alcohol and borax-formaldehyde solutions, and these preparations also proved to be the least corrosive.

Soaking knives which have been used to lance pus pockets in "70 per cent rubbing alcohol" for one hour is not a method to be recommended, since it is doubtful whether the spores present are killed by such a procedure. Acetone is a relatively weak germicide, even against nonsporulating organisms, and there is no reason to believe that even prolonged submersion in this agent would contribute much to sterilization of contaminated instruments. As far as can be ascertained, the proposed mixture of acetone and isopropyl alcohol has not been investigated for its antibacterial action; but, since neither of the ingredients is very effective against spores when tested independently, one would not expect much from the combination.

The fact is, no single solution has been proposed as yet which combines the virtues of noncorrosiveness, reasonably rapid antibacterial activity and volatility sufficient for practical air drying without residue. Exposure to steam under pressure remains the most reliable and satisfactory method of sterilizing contaminated instruments. If "cold sterilization" is to be used, at least four steps are required: (1) thorough mechanical cleansing of the instruments so as to remove all blood, pus and other foreign material which might harbor bacteria, (2) prolonged soaking in a formaldehyde-alcohol or borax-formaldehyde solution, (3) rinsing with water and (4) drying under sterile conditions. There is no reason why acetone should not be used to facilitate this last step. Syringes and needles dried with acetone can safely be used for intravenous work, provided they are sterile.

LAUGHING AND PETIT MAL

To the Editor:—A man aged 36 complains of drowsiness and petit mal attacks brought on by any humorous situation. He loses consciousness momentarily but usually recovers before he falls. Things go black, yet he is aware of his surroundings to some extent. He can control his attacks to a certain extent by refraining from laughing. A distant relative is believed to have reacted similarly. She got relief from thyroid therapy. His neurologic examination is negative. He has ruddy cheeks, weighs 155 pounds (70 Kg.) and is 5 feet 6 inches (168 cm.) tall. The thyroid is normal. He has no other symptoms of thyroid insufficiency. The basal metabolic rate is —20. His temperature averages 97.6 to 98.2 F. The blood pressure is 105 systolic and 60 diastolic. There has been no response at all to 10 grains (0.65 Gm.) of thyroid daily and no ill effects. I have increased it to 16 grains (1 Gm.) daily (Armour's thyroid). Why haven't there been any toxic symptoms? How much thyroid can I give? Do you think this is the wrong therapy? Would you advise amphetamine for the drowsiness and phenytoin for the petit mal? Any advice will be appreciated.

C. L. Feiler, M.D., LaFayette, Calif.

Answer.—The diagnosis of petit mal should be confirmed by an electroencephalogram. Laboratories for such examinations are now being established in various parts of the country. The literature on petit mal and electroencephalographic records is summarized by W. G. Lennox (Science and Seizures, New York: Harper & Brothers, 1941). As Lennox states, "the electrical record of the brain gives the most dependable evidence about the type and severity of the condition, the effect of treatment and the hereditary implications" (p. 93).

Prodromes of various types are frequently found in the period preceding a paroxysm. Gaiety and laughter are among them, and Dostoievsky, the Russian novelist, according to S. A. K. Wilson (Neurology, London, Edward Arnold & Co., 1940) had periods of "supreme ecstasy" before attacks. Thyroid treatment is not known to have any effect on petit mal.

Treatment should consist of phenobarbital or phenytoin sodium or both. The combination of these two drugs not infrequently is better than either given by itself. The use of amphetamine is not advised. The dose of phenobarbital and phenytoin sodium is gaged by trial, although if repeated electroencephalograms can be taken at intervals of three months a more exact evaluation of the amount of drug needed to control the attacks can be estimated.

The dose of thyroid is excessive and certainly carries dangerous potentialities. It is always advisable when administering thyroid to check carefully for heart rate, tremor and basal metabolic rate at frequent intervals. Furthermore, thyroid in this instance does not seem indicated.

GENERALIZED RASH AND PREMATURE DELIVERY

To the Editor:—A woman aged 34 had three previous pregnancies. During each pregnancy there was a rash with considerable itching all over her body in the fourth month. Each pregnancy terminated prematurely at the beginning of the seventh month. The rash disappeared with the termination of pregnancy. Only one child survived. She is now pregnant again and I should like to prevent the rash and the premature delivery. The previous three deliveries took place out of town and I have only scanty information about them. The physical examination was negative with the exception of an inclination to increased blood pressure (145 systolic, 80 diastolic. The blood Wassermann reaction was negative; the urine examination was negative; the blood count, including a differential count, was normal. The urea nitrogen was within normal limits. The oxygenads were normal. I have the feeling that we have to deal in this case with an allergic reaction toward some products of pregnancy. Is there any possibility that the patient may be desensitized? Is progesterone indicated in this case?

Joseph Kaschmann, M.D., Hartford, Conn.

Answer.—The generalized rash which develops late in pregnancy is probably toxic in origin but it may be on an allergic basis. There have been no attempts at desensitizing such a patient, probably because the complication is so rare. Generalized cutaneous eruptions may be on the basis of a neurodermatitis, particularly in high strung, nervous persons. These patients should be treated with sedatives, such as the barbiturates, plenty of rest and a carefully selected diet. Locally, soothing antipruritic lotions usually suffice to give the patient relief.

The premature termination of pregnancy in this case may well be due to a latent vascular-renal disease which is not apparent in the usual examination. Other factors may play a role in such cases. Abnormalities of the reproductive tract or an abnormal endocrine balance occasionally accounts for such a course. The patient should have a basal metabolism test and, if the reading is below normal, thyroid in liberal amounts should be administered throughout the pregnancy. The use of vitamin E in the form of alpha or mixed tocopherols has been advocated. The latter can be given in 40 or 50 mg. capsules two or three times a day during the entire pregnancy. Progesterone is of value if it is administered during the first part of the pregnancy. One mg. parenterally can be given three times a week. Progesterone is likewise available as a salt which is efficacious by the oral route. Some success has been reported in the prolongation of pregnancy by the administration of this material during the last two or three months of the gestation. Unusual stimuli such as coitus, douches or violent exercise should be omitted during the last trimester.

POLLENS IN BRAZIL

To the Editor:—In order that I may properly advise a patient who is leaving for Rio de Janeiro, will you please publish what data are available relative to the pollen count in that area?

Allen L. Richardson, M.D., Detroit.

Answer.—Data have not been published on pollen or fungus spore incidence in the immediate vicinity of Rio de Janeiro. However, daily studies were made at Belo Horizonte, some 175 miles north of Rio de Janeiro, by J. B. Greco, A. Oliveira Lima and A. Tupinambá from January 1940 until July 15, 1941. These investigators found little pollen from trees or weeds of any kind, although small amounts of Western ragweed (Ambrosia psilostachya) are present in the area. In their article awaiting publication in the *Journal of Allergy* exact figures are not given, but they note a definite grass season lasting approximately from the middle of May until the middle of June. The pollen at that season is attributed principally to Bermuda grass (Cynodon dactylon).